BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

Vol. 33.] 1936. [No. 12.

PELLAGRA AND PELLAGRA-LIKE CONDITIONS IN WARM CLIMATES.

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Section III.*

Having discussed some of the less commonly recognized features in the symptomatology of pellagra in the last Section, I shall now deal with a number of conditions, variously described by authors, which I believe are pellagrous in nature or are closely related to pellagra.

As mentioned above, glossitis is a common symptom in pellagra and one which may antedate those upon which a diagnosis is made, by months or years. In 1925 and in 1930 I drew attention to the outbreaks of epidemic glossitis among natives, described by observers in Africa and elsewhere and suggested their probable relationship with pellagra. They will be briefly referred to again here.

H. Jamin (1925) described from Tunis what he called "stomatite d'automne," an affection coming under observation each autumn, in which the patient complains of a sensation of heat and tingling in the buccal mucosa. The chief lesion consists in a desquamatory glossitis with similar changes on the gums, lips, cheeks, and soft palate.

The same year M. Nogue (1925) published an account of epidemic glossitis in December at Dakar, Senegal, occurring chiefly among native children. A characteristic denudation of the tongue, generally affecting the anterior third, was seen, often associated with similar lesions on the inner surfaces of the lips and cheeks and excoriations at the angles of the mouth, giving rise to a macerated appearance. This condition caused a burning sensation in the mouth on eating. In some outbreaks gastro-intestinal symptoms were also noted and it was observed that both tended to clear up together on improved feeding. It was suggested that the ingestion of peppermint and salt-fish played a part, but it is more important to remark the seasonal incidence of the condition.

C. Mathis & Guillet (1925), who had the opportunity of seeing the cases described by Nogue, add fissuring and hypertrophy of the papillae of the tongue to the picture. They attempted to demonstrate

* Section I appeared in the October issue (pp. 729–741) and Section II in the November issue (pp. 815–825).

a causative organism but no positive evidence was advanced and the condition remained unexplained.

J. MONTPELLIER, A. CATANEI & L. COLONIEU (1927) have published observations on an affection, differing little from the above, met with in Algiers, but gastro-intestinal symptoms and seasonal variation were not noted.

A rather similar condition was described by J. KATZENELLENBOGEN (1928) in Palestine as one of the most common and distressing complaints among labourers living on a monotonous diet chiefly of preserved food and legumes. The malady was seen in the months December to February and on grounds which would appear entirely unconvincing was considered as due to the pneumococcus. The glossitis was accompanied by inflammation of the lips, soreness at the angles of the mouth and in many cases the throat was also involved. After some six to eight weeks the condition improved.

M. G. BLACKLOCK (1925) remarked what would appear to be this same form of glossitis among many of the school children of Sierra Leone which gave rise to local discomfort but no other symptoms.

None of these authors has demonstrated any cause for these conditions and at the same time none has suggested the possible link with pellagra.

These outbreaks of epidemic glossitis with a seasonal incidence should be compared with (*a*) the condition described by J. T. BRADLEY (1929) among the inhabitants of the outlying islands of Seychelles, called " décoquée " in which soreness at the angles of the mouth, soreness and redness of the eyelids, erythematous rash on the genitalia, scrotum or vulva, were associated with variable knee-jerk and sometimes affections of vision and hearing. It was noted that sufferers improved if they went to the mainland and that those in a position to obtain a better than the average diet did not suffer.

(*b*) The " Outbreak of Exfoliative Glossitis in an Assam Jail " published by G. H. FITZGERALD (1932), and (*c*) the condition described as " The A and B avitaminosis disease of Sierra Leone " by E. J. WRIGHT (1928) and (1930).

Under the designation Exfoliative Glossitis G. H. FITZGERALD described an outbreak of disease in one-third of the inmates of the Gauhati Jail, Assam, consisting of a glossitis in which progressive denudation occurred, with the production of a large, indented, fissured, red-glazed tongue with enlarged red papillae, associated with a generalized stomatitis, salivation and fissuring at the angles of the mouth with heaped-up white sodden epithelium.

Gastro-intestinal symptoms occurred in 37 per cent. of cases; flatulence and eructations, abdominal pain about the umbilicus and oppression after food. Tingling in the hands and feet was noted in 2·75 per cent., a pellagrous-like eruption on the dorsa of the hands and feet occurred in 2 per cent. There was no diarrhoea but a previous history of " Flexner " infections was obtained in 30 to 40 per cent. of those affected.

The outbreak occurred in the autumn of 1931 and the cases were watched only till April 1932. In March an attempt was made to see the effect of treatment by diet. Improvement took place in 70 per cent. of those from whose diet certain irritative elements were eliminated (chillies); in 70 per cent. of those to whom 2 drachms of cod-liver oil were administered daily, while improvement in 92 per cent. was seen

in those to whom cod-liver oil and 1 ounce of yeast were given, and in 93·75 per cent. of those to whom yeast alone was administered.

After improvement had been thus shown with a few weeks' treatment, all groups were placed on the standard diet with condiments; active lesions again appeared in 22 per cent.

The jail diet consisted of:—Rice 12 chittaks, salt 7/16, dal $2\frac{1}{2}$, vegetables 3, mustard oil $\frac{1}{4}$, condiments $\frac{1}{8}$, antiscorbutics $\frac{1}{8}$.

The affection described by WRIGHT in Sierra Leone was observed throughout the year but had a marked seasonal predominance towards the end of the rainy season, when there is an annual shortage of food. The symptoms in order of appearance had reference to the tongue, the angles of the mouth, eyes, nose, anus, scrotum, and vulva, and the nervous system. The first named symptoms are identical with those described in pellagra by STANNUS (1912 and 1913). In addition the texture of the skin of the limbs and trunk in Wright's cases was altered by a keratosis which caused it to be dry and rough. The nervous symptoms included variable knee-jerk, tongue tremor, sensations of tingling, heat, cold or numbness, impaired vision and hearing, later Rombergism, inco-ordination, ataxia, paresis of limbs.

There is much to suggest that the affections described above under the term "Glossitis" are, in fact, examples of early pellagra, or rather what has been termed prepellagra as mentioned in a previous section. Together with "décoquée" they form a series of conditions leading up to that described by FITZGERALD, which would be recognized I think by any pellagrologist as pellagra. In commenting upon the affection met with by WRIGHT in Sierra Leone STANNUS (1930) remarked "with very little hesitation one may suggest that his cases are cases of pellagra."

In attempting a differential diagnosis from that disease, WRIGHT says "pellagra with which the disease might be confused is commonly found in the white races"—a remark somewhat beside the point. Again he says "In pellagra (*cf.* syn. Alpine Scurvy) there is a condition of the gums similar to that produced by a vitamin C deficient diet"—showing thereby some misconception upon the point. Further, in a table of differential diagnosis, Wright states that wasting, effusions and oedema, salivation, pyrexia, painful deglutition, irritability and insanity are present in pellagra but absent in the Sierra Leone disease—thus failing to realize, I think, that pellagra may present a very varying picture and that all or none of these signs may be present in that disease. Further he states that deafness, diminished skin sensibility, foot- and wrist-drop which he finds in Sierra Leone do not occur in pellagra, a statement which needs some modification. Lastly in regard to the skin WRIGHT appears to think that in pellagra the lesion is always of the nature of an acute erythema on exposed areas only and that keratosis does not occur, thereby exhibiting a disregard for what is known about the skin in pellagra. Enough has been said to suggest that this attempt at differential diagnosis breaks down when subjected to careful scrutiny. The Sierra Leone disease is, in my opinion, pellagra in all possibility.

FITZGERALD believed that a diagnosis of an early pellagrous condition seemed a probable one in his cases. There can be little doubt he was correct in his surmise, in spite of the fact that skin lesions comparable to those of pellagra were only noted in two cases. I would add that probably the irritative elements in the diet served to determine the mouth as the chief site of the pellagrous exanthem.

One of the more recent communications upon these pellagra-like conditions is that by J. V. LANDOR & R. A. PALLISTER (1935) describing an affection characterized by what they call " eczema " of the scrotum, " eczema " of the angles of the mouth and superficial glossitis, followed later by symptoms of combined degeneration of the cord and poor vision, occurring among the inmates of the prison at Johore Bahru and due, in their opinion, to an avitaminosis B_2.

On 29th August, 1933, among a total of 393 prisoners, 138 had stomatitis and the affection of the scrotum, 59 stomatitis only, 8 scrotal affection only. The free margin of the prepuce in some cases and more rarely the anus exhibited changes similar to those at the angles of the mouth. The descriptions and photographs demonstrate clearly that these conditions are the exact counterpart of those described by STANNUS in African pellagrins, though in many of these Johore cases the scrotal condition appears to have been complicated by a tinea infection. About 2 or 3 months after entering the prison nearly 50 per cent. of prisoners developed some or all of these symptoms. The " attack " lasted a few months and was unaffected by an experimental change in diet. In earlier cases, if looked for, pain, tingling, " pins and needles," and numbness in the lower extremities, haziness of vision, and increase of knee-jerk, were discovered. Later pains, stiffness, and weakness in the legs were complained of. Rombergism and an ataxic gait developed and sensibility to touch, pain and deep pressure in the leg, arm and abdomen were affected, even sometimes the face. Vision might be 6/60 but complete blindness was never seen. The pupils reacted, but light reaction was sometimes poorly sustained. No case of night-blindness or xerosis was observed. Mentally some of the patients were dull.

All those showing nervous symptoms had been in the prison over a year. In Johore 9 of 140 prisoners who had been in prison a year, of the total of 393, showed nervous system affection. In Singapore the figures were 34 ; 461 ; 1,210. Those working as cooks appeared to escape the disease. The malady is said to have been noticed in Johore prison for 30 years past. It may also be seen in the poorer members of the outside population. Among prisoners, Chinese, Malays, Indians and one European have all been affected. The affection has also been noted in the Kuala Lumpur Prison, the Mental Hospital at Singapore and among the boys of the Singapore Reformatory.

Investigation showed no more than a mild anaemia in some cases. Four of 17 cases showed achlorhydria.

The results of treatment were interesting : 50 early cases were " cured " after 15 gm. marmite daily for 1 month, 20 relapsed within a month of ceasing to take it ! Cod-liver oil and orange juice had no beneficial effect. Four ounces fresh yeast daily cured most cases as did autoclaved yeast, but the best results were obtained by giving half a pound of liver daily. The authors say " The relationship of the disease described and classical pellagra is interesting but a little obscure." Pellagra does not occur in Malayan prisons though sporadic cases do occur in the general population and three such cases are cited—a hawker, a fisherman and a ricksha-puller. All had, besides the classical exanthem, diarrhoea. The hawker had a stricture of the rectum, the fisherman rectal ulceration. The ricksha man had the affection of the lips and scrotum as seen in the prisoners.

Again, the pellagrologist would have no hesitation, I think, in pronouncing this condition described by LANDOR & PALLISTER to be

pellagrous. In this series of cases the high incidence of the pellagrous scrotal skin affection was quite possibly determined by the tinea infection. One wonders if in these cases some other manifestations of pellagra would not have been detected by the trained eye ; the hesitation to make the diagnosis of pellagra has been due apparently to the absence of the classical exanthem.

P. MANSON-BAHR (1935) in a letter referring to this paper by LANDOR & PALLISTER and their observations upon the lips, tongue and angles of the mouth, states that he noted the same conditions present in 181 of 1,461 individuals examined in Ceylon (when studying sprue) among Sinhalese, Tamils, Japanese and Malays alike. The condition was not associated with diarrhoea ; at that time he thought it was due to eating dried fish.

D. G. Fitzgerald MOORE (1934) has described a condition of considerable interest among native school children in Nigeria. While 12 per cent. of the boys, who were boarders and poorly fed, suffered from the affection, no cases occurred among those that lived at home and none among the girls who were taught cooking. In all some 300 cases were seen in five years. Some cases were also seen among the poorer ill-fed casual labourers who lived on cassava, dried-fish, oil, suet, pepper. At the beginning there was a sore tongue and mouth with aphthous lips ; a dry scaly itching condition of the scrotum or vulval irritation supervened, followed later by dimness of vision and loss of central acuity, but unaffected fields. In these cases a diagnosis of retrobulbar neuritis was made and later pallor of the temporal halves of the discs was seen. Some patients complained of epigastric pain and achlorhydria might be present. The diet of these schoolboys consisted of one cabin biscuit and cassava soup—" gari," with only 3 oz. of meat a week, practically no protein and much of the cassava was bad.

During the holidays the condition got better or in early cases treatment with marmite, oleum morrhuae, iron and " tombo "—a fermented palm sap—yielded good results. The author notes the resemblance of these cases to Wright's cases in Sierra Leone but in one there was retrobulbar neuritis, in the other group the cornea was affected.

He believes there is a dietary deficiency involved, but also that " gari " acts as a toxin.

The interest of this group of cases is obvious, linking up as it were, some of those considered above. Once more, I should hazard the diagnosis of pellagra in the Nigerian school-children.

The next communication of interest is by L. NICHOLLS (1933 and 1934). He describes a syndrome which he found to be common among the inmates of a gaol in Ceylon consisting of a dry furfuraceous skin and a rash consisting of enlarged sebaceous glands plugged with altered sebum, which may become surrounded by a darkly pigmented areola. This rash has a characteristic distribution on the thighs, the external surfaces of the arms near the elbows, the abdomen and across the shoulders, and occurs less commonly on the face, neck and chest, as is well shown in his photographs. The associated symptoms are dimness of vision, a " neuritis " resembling that of pellagra, " burning sensations of the hands and sometimes of the feet," numbness and tingling of the legs, weakness, xerophthalmia, and often death. Diarrhoea was common, possibly due to " Flexner " infections. Most of these prisoners came from an indigent class among whom dry skin and keratomalacia are not uncommon. To the skin condition he gives the name " Phrynoderma "—toad skin.

Later L. NICHOLLS (1935) refers to the bewildering variety of clinical nervous symptoms met with in the cases mentioned above and notes their resemblance to those seen in children and in women towards the end of pregnancy or during lactation, well and long recognized in Ceylon. They include burning of the palms and sometimes of the soles, tingling, numbness and weakness, often later mild paresis, ataxia, and rombergism, loss of joint sense, inco-ordination, coarse tremor and at this stage loss of deep reflexes. In children phrynoderma is associated with emaciation and keratomalacia. Examinations of the central nervous system of such children post-mortem showed many degenerate fibres in the posterior nerve roots and posterior columns with scattered degeneration in the lateral and anterior columns, and in the anterior roots.

These cases of NICHOLLS' are examples, one might suggest, of chronic pellagra. The condition he calls phrynoderma is no new condition—it has been described by many other writers on pellagra as mentioned in Section 2. His reference to "the bewildering variety of clinical nervous symptoms" is peculiarly interesting as this is particularly true of pellagra.

Of interest too is a reference by L. NICHOLLS (1933) to a condition he had observed many years before among indentured native African labourers which affected them within a few weeks of their arrival as healthy individuals at the Magadi Soda Factory, East Africa. It was characterized by a dry goose-skin-like condition due to an enlargement of the sebaceous glands into papules together with a furfuraceous desquamation. Associated with this condition there was what he called a mild form of neuritis; later diarrhoea supervened and many deaths ensued. NICHOLLS says that a similar condition occurred in the Native Carrier Corps in East Africa during the War.

These conditions, in my opinion, might well be pellagrous, though not recognized as such.

Now NICHOLLS was acquainted with pellagra many years before in the West Indies but I believe at that time no diagnosis of pellagra was ever made in the absence of the text-book trinity of symptoms, and perhaps for this reason he has failed to identify the Ceylon affection with that disease.

We owe another description of this pilo-sebaceous folliculitis to L. J. A. LOEWENTHAL (1933). He described a skin condition among the inmates of the Uganda Central Prison which he considered was due to a vitamin A deficiency. He thought it had not been previously recorded, the only account of any condition resembling it, which he knew of, being one by PILLAT, though in his opinion the two were not identical. One hundred and thirty cases were found in the prison at Mulago, most of whom also exhibited xerophthalmia and night-blindness. Similar cases were later discovered in the prison at Nairobi when sought for.

The lesions he describes under several headings:—(*a*) general dryness of the skin of the body (but not of the face), which becomes rough and grey-black in colour, the greyness depending on the desquamation. It is most marked on the backs of the hands, the front of the legs, both surfaces of the thighs and across the buttocks, and causes itching. (*b*) Smooth-topped, shiny papules, distributed over the exterior surfaces of the arms, the anterior and external surfaces of the thighs and sometimes on the buttocks, loins, chest and back. (*c*) A folliculitis in which the mouths of the pilo-sebaceous follicles are sealed

with a mass of horny tissue accompanied by hyperkeratosis in the immediate neighbourhood and deep black pigmentation. (*d*) Acne. As the cases clear up the contents of the follicle are extruded leaving a black macule. The diet consisted of maize 20 oz., beans 5, dry meat 2, nut 3, suet ½, sweet potato 10 oz. The cases quickly improved with the exhibition of vitamin A-containing substances, *viz.*, one ounce of cod-liver oil.

LOEWENTHAL has given a perfect account of the pilo-sebaceous lesions as they occur in pellagra; the association with the dry rough skin condition, as described, further links these cases up with that disease. The resemblance of this condition to that recounted by NICHOLLS is obvious, further made good by xerophthalmia in both sets of cases. In the Ceylon cases, however, there were added nervous manifestations.

I must here digress for the moment to discuss what, for short, I will call the "folliculitis" as above described.

G. P. GOODWIN (1934) has described the same condition in a child aged 10 in a London hospital as a "cutaneous manifestation of vitamin A deficiency." Associated with the "folliculitis" (I was very kindly given the opportunity of seeing the boy) there was dryness of the skin, particularly noticeable over the legs and feet; the nails of the fingers and toes looked drier than normal; "a small area of moist eczema was present at the left angle of the mouth"; injection of the cornea was noted; "the tongue was red and smooth being denuded of its superficial papillae"; the gums were swollen; no xerosis, no gastro-intestinal symptoms. Dr. Helen MACKAY (1934) in reviewing the subject of "vitamin A deficiency in childhood" suggests that in more advanced cases, to the above-mentioned symptoms might be added periods of diarrhoea, emaciation, scaly pigmentation followed by a shrivelled condition of the skin, with powdery or flaky desquamation, xerosis and mental apathy—a condition which, leaving out the question of causation, I suggest might well be included under the designation of pellagra.

A. PILLAT (1929) writing from Peiping in an article on keratomalacia among the Chinese includes under this heading xerosis conjunctivae and xerosis corneae as well as keratomalacia proper. He finds that night-blindness may be found in these patients and that besides these are many other associated signs including a pale grey colouring of the skin of the face, neck and forearms, which later becomes yellowish. The skin may be puffy and the face look bloated. The skin becomes covered with "a remarkable quantity of scurf," like "powdered flour," and there is an affection of the sebaceous glands, causing large numbers of comedones on the face, the secretion remains in the ducts and the skin about them becomes black. The skin is dry and rough, due to hyperkeratosis; in some cases ulceration occurs, referred to by PILLAT as Dermomalacia. The hair is dry and falls; the nails become dry, furrowed and white. There is decrease in hydrochloric acid secretion, diarrhoea, hoarse voice, bronchitis, and a genito-urinary affection all of similar nature, *i.e.*, a disturbance in the nutrition of epithelial cells, due, he thinks, to a vitamin A deficiency.

The resemblance of LOEWENTHAL's cases to the condition described by PILLAT and of both to pellagra is obvious.

Another recent paper is one by J. HARKNESS (1935). He has described what he considered to be a deficiency disease among natives of the Bukoba district of Tanganyika Territory whose diet consisted chiefly of cooked bananas, small quantities of ground-nuts, and beans, and but very rarely fish, meat or milk.

The chief symptoms were night-blindness, with ulceration of the cornea in some cases, and chronic enlargement of the parotid glands. The tongue was sore, glazed, and tremulous or coated in the middle and red at the margins with denudation, the gums were also sore. " Dermatitis is seen, and in the early case the skin is slightly swollen and the seat of burning and itching. A butterfly patch over the nose and molar regions is present." Casal's necklace is more rarely to be observed. The hair of the outer side of the eyebrow is lost and the nails are brittle and cracked. " In later stages the skin is thick and rough, especially on the elbows and backs of the hands . . . the patient becomes dull, stupid, and subject to dementia. Intractable diarrhoea, emaciation and death in advanced cases occur." Profound atrophy of the intestinal mucosa was found post-mortem. The author believes the condition to be pellagra, and there can be little hesitation in agreeing with him.

In a *Contribution à l'étude des syndromes d'avitaminose* A. DUMONT (1934) cites cases met with among native labourers in the Bas-Congo. He appears to believe that in beriberi there are two syndromes, the one comprising the cardiac condition and oedema due to shortage of vitamin B, the other including the neurological manifestations due to toxi-infections by various organisms which come into play as a result of gastro-intestinal disturbance. Among his cases, however, are a number exhibiting symptoms very reminiscent of pellagra rather than beriberi ; conjunctivitis, blepharitis, a plaque of hyperkeratosis under each eye, a rhagades-like condition at the palpebral and buccal commissures, infiltrated tongue, gingivitis, the mucous membrane being depolished and covered with a grey adherent pellicle. There was no affection of the cornea. He speaks of the " syndrome oculo-buccal," and thinks it has affinities with scurvy. It clears up upon an hypervitaminic diet.

The last of these recent papers which I shall include here is that by N. L. CORKILL (1934) concerning pellagra among a small group of millet-eating people living to the N.E. of Khartoum, Sudan. Pellagra among millet-eaters in Egypt has been recorded by W. H. WILSON (1921) but only two cases of the disease had been previously noted in the annual Reports of the Sudan Medical Service. Although the diagnosis of pellagra was made by CORKILL his cases are included here as the diagnosis rested upon all the less common manifestations of the disease to which I have drawn attention and no case of frank " text-book " pellagra was seen. The history of the discovery of the malady in the Butana country by CORKILL is well worth being read as an example of how illusive pellagra may be. The data were collected during two visits to the district of only a few days and many of the facts gradually emerged in conversation with the people.

Putting two and two together he found a group of symptoms which included weakness, wasting, flatulent dyspepsia, a history of diarrhoea, " burning pains " in the extremities (almost diagnostic—it occurred in nearly 90 per cent. female and 80 per cent. male cases), pains in the body, head and limbs, vertigo, abnormal knee-jerks, mental symptoms, etc. There was a history that night-blindness was common among these cases. There was also a history of burning pain in the skin followed by swelling, dermatitis, desquamation, and pigmentation. Pilo-sebaceous gland dysfunction, powdery desquamation and " sulphur flake " were noted. Typical glossitis, stomatitis, pigmentation of the gums and angular stomatitis were found to be present, but

there is no mention of the similar conditions of the prepuce, anus and vulva, nor of any affection of the scrotum, probably because the necessary examination was not carried out. Finally there was a seasonal incidence associated with the symptoms.

Lastly reference must be made to H. STRACHAN's observations in Jamaica upon a series of cases, among which there were undoubtedly some of pellagra, as I have previously pointed out. At the time he ascribed the malady which he recorded as due to malaria, though he remarked that there were many features suggestive of beriberi. His observations were first noted in *Sajous's Annual* (1888), his paper later appearing in the *Practitioner* (1897). Among the symptoms were "burning pains in the palms and soles," numbness and cramps in the hands and feet; the palms and soles showed hyperaemia and fine desquamation and became more and more deeply pigmented as might the whole body; there was branny desquamation at the margins of the eyelids, lips and nostrils; the corners of the mouth, the prepuce, anus and vulva were the seats of excoriation; hyperaemia of the conjunctiva and of the mucous membrane of the mouth with loss of surface epithelium of the tongue were noted; emaciation was marked and a number of deaths occurred.

There were besides, references to an odd medley of nervous symptoms; some of these suggest pellagra, but H. H. SCOTT, criticizing Strachan's reports, thinks that he without doubt included under the one heading of Peripheral Neuritis a variety of cases of all kinds.

In 1930 I referred to SCOTT's interesting description of an acute outbreak of "central neuritis" in Jamaica and to his observations upon the resemblance of the nervous lesions found to those in pellagra, though he thought the evidence was against the condition being acute pellagra or any other deficiency disease. He rather believed this Jamaican outbreak was due to an intoxication, the toxin entering by the mouth. It is just worthy of note, however, that the epidemic occurred among adult native labourers, male and female, on sugar estates during the cutting of the cane crop, and ceased when this operation was over. The onset was very acute, 50 to 100 new cases occurring per day.

The first symptom was always a conjunctivitis with swelling and redness, photophobia, ulcers and abrasions of the lids. The second symptom was burning of the mouth, which was red, inflamed, and the site of aphthous ulceration with fissuring at the angles of the lips. The tongue was not sore as a rule and salivation not common. Some two weeks later either (1) diarrhoea set in, from which recovery might take place spontaneously without further symptoms or from which the patient died, or (2) constipation with nervous symptoms developed, consisting of "burning in the toes and soles of the feet," a feeling of numbness and tingling, spreading up the leg causing inco-ordination and difficulty in walking but no loss of power or sensation, absent knee-jerk but no wasting. The upper limbs might be similarly affected and later there might be diminution of visual and auditory acuity and a peculiar steppage gait which remained permanently. The ordinary diet of these people was deficient in protein but contained an excess of carbohydrate and consisted of yam, bread-fruit, cocoa, peas, beans, corn-meal, salt-fish, etc., but during the harvesting the workers lived almost entirely on sugar cane.

"Burning pains" in the soles of the feet and in the palms of the hands is a symptom so constantly recurring in the descriptions of the

symptomatology of pellagra that I have come to consider it as one of considerable importance and great diagnostic value. The non-mention of this symptom in some accounts I believe does not necessarily mean that it was not present, as in many cases only such signs are given as to warrant a diagnosis of pellagra. It will have been noticed in discussing the pellagra-like conditions above how often this symptom has been noted.

In the pages which follow I have tried to give the history of the recognition of this manifestation as it has been described as a separate symptom.

On November 5th, 1825, J. GRIERSON, Esq. (1826), attached to the Arracan division of the army, presented a paper before the Medical and Physical Society of Calcutta entitled "On the Burning Feet of Natives." "It exists," he says, "in various degrees of severity, from an uneasy harassing sensation of heat and tingling to the painful extreme of burning, destructive of sleep and appetite in the first instance and latterly of serious injury to the general health." The affection may also include the palms of the hands, aching along the tibiae is a common accompaniment, and giddiness may supervene. The condition may be unconnected with other troubles or may be associated with "febrile and bowel complaints."

Grierson did not know of any previous observations on the malady but refers to an affection of the soles of the feet among Laplanders, for which the bark of the beech is used "as a remedy for unpleasant symptoms resulting from checked perspiration."

In a footnote to this paper there are some remarks concerning treatment of the condition by Mr. PLAYFAIR (1826).

"Burning of the feet" was described by Assistant-Surgeon MALCOMSON of the Madras Medical Establishment (1835) as occurring in Madras sepoys at Ava during the Burmese War, when subjected to fatigue and exposure, on a diet consisting of rice, 2 oz. ghee, a little salt-fish and spices, and was differentiated from beriberi.

WARING (1860) in an article under this same heading, states, almost unknown before the first Burmese War in 1825, it is an affection much more commonly seen in Arracan, Burmah, the Tenasserine Provinces, Penang, Singapore and other eastern settlements and less commonly in India. The symptom of burning feet may be the only one present but "most generally appears as a sequel of fever, bowel complaints, rheumatism and beriberi and is not infrequently concomitant with some form of leprosy." The mention of leprosy is interesting as it seems possible that a pellagrous eruption was here mistaken for leprosy. This author quotes MALCOMSON as considering the affection to be "connected with scorbutic disease," he also points out that the palms of the hands may be similarly affected. The anterior part of the foot is chiefly the seat of the pain, the instep and heel less, according to GRIERSON.

WARING also quotes MCKENNA "The precursory symptoms . . . languor, loss of appetite; pain in extremities . . . shooting over body . . . more acute in larger joints; countenance anxious and cachectic . . . bowels usually regular, sometimes seized with looseness: debility, irritability, sleepless nights, emaciation: burning and pricking at the soles of the feet and palms of the hands, in due course diffused over the whole body with exacerbations by day and remissions in evening with sundown: exhaustion and sometimes death."

Playfair is also quoted as pointing out that there are two types of the malady—in the one sweats occur, in the other the skin is dry and scaly; again a significant observation.

Billod cites Hurst as saying that "Burning Feet" among Cipayes (Sepoys?) was attributable to the use of damaged rice.

J. N. Dugdale (1928) writing upon "Burning Feet" in Estate Medical Practice in Malay found the condition most commonly in middle-aged, ill-nourished coolies who had been in the country months rather than years. There was no redness and rarely swelling but sometimes engorgement of blood-vessels. There was an associated peripheral neuritis (*sic*) with wasting and rombergism. Sir David Galloway is quoted as having been familiar with the condition for 40 years, and Dr. Wilson knew of it in Johore. In correspondence A. B. J. Coope (1928) stated that in Johore the condition was confined to Tamil coolies, and associated with night-blindness and evidence of scurvy—swollen gums, emaciation, harsh dry scaly skin, variation in pigmentation and cracks in the lips. The affection occurred in periodic waves at times of economic stress; it responded to antiscorbutic treatment. Assistant-Surgeon W. Ray (1902) alludes to a few cases of pellagra in N. Behar in which "burning of the skin of the feet and hands" was a prominent symptom, associated with reddish areas of skin, roughness and dryness; the tongue was red and dry, and diarrhoea was present but no nervous symptoms were noted. W. B. Morgan (1929) refers to the condition—"a deep seated burning sensation" in the soles of the feet—as common among Tamil emigrants to Malay, recruited from S.W. Madras but not among Telegus who take a better diet. The symptom appears after twelve months' residence in Malay and is associated with insomnia, mental depression and nostalgia. "More than half of these patients showed small black cutaneous nodules over the external surface of the legs and forearms." Nitroglycerine was given on the supposition that there was over-stimulation of the sympathetic nerves due to a deficiency of vitamin B group, and a diet of maize-raggi, eggs and mutton. Cure is said to have been thus effected in two weeks. This affection is seen in Singapore, Deccan and in Ceylon, but not in Java and Sumatra, it is stated.

Brahmachari (1923), referring to an outbreak of epidemic dropsy in 1919–1920 at Krishnagar when 127 out of a total of 171 cases came under observation, places "burning pain" in the soles and palms as among the most common symptoms. It was associated with pains in the calves of the legs, anorexia, diarrhoea, weakness, anaemia, and oedema. The knee-jerks were normal in 90 per cent. of cases. No mention was made of skin lesions.

J. C. R. Buchanan (1932) has recorded interesting observations upon a condition which attracted his attention "by the universality of its occurrence in Somaliland" among the natives, one of the chief symptoms being "burning feet." One hundred cases were analysed: Pain of rheumatic type was almost universal—general aches; pain in joints, bones and muscles; headache and backache; tender shins. "Burning feet" and itching or tingling of the skin also constituted a characteristic symptom in 39 per cent. "Burning palms" was noted in 3 only of the 100 cases. Oedema, in the vast majority of cases confined to the legs, occurred in a large proportion. Severe pyorrhoea was very general among them and epigastric pain occurred in 45 per cent.— a dull heavy pain, worse after food, but the appetite was not affected; constipation was the rule; the urine showed no albumin. In 15 cases

there was some myocardial involvement. Some of the ordinary signs of pellagra such as skin changes, soreness of the tongue and lips, diarrhoea and psychosis were noted among these people.

This disease, if it can be so called, is a very chronic one; "it may go on for ten years without producing much more than slight disability—and would appear to be seldom if ever fatal." The condition occurs among various groups, jungle native and townsman, on rather different diets, but the author remarks "It can be said that these diets contain most of the essential elements but that they lack variety and balance; it is doubtful if the vitamin content is always adequate." The margin of safety is small, it is said, but it is noteworthy that in the several group-diets meat and milk appear, though fresh vegetables are completely absent. The results of treatment were interesting: Of 22 cases treated by intravenous injections of 10–25 minims of tinct. iodi. in 4 to 8 cc. distilled water, twice weekly, 19 improved. Of 17 given an addition of yeast and onion to their diet 14 improved. On combined treatment 10 out of 11 improved. The author thinks that the condition resembles the "pre-beriberi" of V. MAHADEVAN & T. K. RAMAN (1930). This seems rather doubtful as in these authors' cases the prominent symptoms described as those of larval beriberi consisted of numbness and tingling with areas of anaesthesia of the skin of the lower extremities, diminished or absent knee-jerk and tenderness of the calf muscles. BUCHANAN's cases would appear to have closer affinities with pellagra.

L. R. SHARPLES (1929) has described "Burning Feet" among Hindu Bengalis born in British Guiana and notes that it is not seen in the rest of the population. It is much commoner among the women, and between the ages of 17 and 40. The hands may also be affected, the pain is worst in the tips of the fingers. Patients also complain of "darkness of the eyes," *i.e.*, that their vision is impaired (? night blindness). There may be rombergism and in severer cases some anaesthesia on the dorsa of the feet but no paresis or wasting. No anaemia is found though ankylostome infections are universal. The diet consists of milled polished rice, and flesh is eaten. Symptoms subside on a good mixed diet being given.

These various groups of cases of "Burning pain" in the soles of the feet and palms of the hands, it will be noted, have been described as having other associated symptoms in most cases. Considered together they would appear to have a close similarity to other groups of cases, before mentioned, allied to pellagra.

Reviewing all the conditions which I have suggested are pellagra-like we see a number of affections in which some one or more symptoms are associated to form a series of syndromes, but all the various symptoms in the several syndromes are symptoms met with in pellagra. Some of these affections are obviously frank pellagra, some might be called pre-pellagrous states or larval pellagra.

In some of the syndromes one or two symptoms appear to dominate the picture rather to the exclusion of others, with considerable variation in the several groups, but the linking and overlapping is very remarkable when all are considered in series.

In collecting together in the form of a summary the various conditions which form the subject of this article it has been my purpose in the first place to help towards the better understanding of the symptomatology of pellagra and secondly to stimulate further interest in that disease and pellagra-like affections.

Shortly I hope to publish a reasoned discussion upon the aetiology of the disease and some points in its pathogeny.

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TROPICAL OPHTHALMOLOGY.

A Review of Recent Articles. XXVI.*

Trachoma.—MacCallan[1] defines trachoma as "a specific contagious disease of the conjunctiva in man. It is chronic in nature. It is characterized by a sub-epithelial infiltration of the conjunctiva by a cellular exudate, which spreads to the cornea and to the tarsus. It is followed by cicatricial changes in the affected tissues." The inflammatory cells may cause no obvious follicle formation, but merely induce a widespread subepithelial infiltration which makes the conjunctiva red and velvety. Bleblike excrescences, which emit a gelatinous material, form in the tarsus during the second stage of the disease owing to a blocking of the ducts of the meibomian glands. The conjunctival secretion in the first stage shows no pathognomonic chemical or microscopical changes, and reliance must be placed on the characteristic vascularization of the upper fifth of the cornea, which is detected by the slit-lamp.

Research by Pulvertaft[2] into the nature of the follicles found in the second stage of trachoma has led him to suppose that these are not true follicles, but excrescences due to blockage of the meibomian ducts owing to cicatricial contraction of the tissues. True follicles do not undergo degenerative changes, and their contents cannot be expressed. He found that the histological changes in the disease are comparable with those occurring in many sub-epithelial inflammations—such as chronic superficial glossitis. The sub-epithelial tissues are densely infiltrated with small round cells—mainly lymphocytes, with some plasma cells : the pathological lesions are in no way distinctive, but are merely the familiar ones of inflammation and incomplete repair.

Marcus & Wainer[3] have studied the varieties of cells found in trachomatous material. After reviewing the observations of other pathologists they describe their own technique. The smears are made from the retrotarsal fold without using much pressure, and after drying, are fixed in ethyl alcohol. Giemsa's stain (1–2 drops per cc. of distilled water) is used to stain this preparation from 45 to 60 minutes. Eosin-azur II is also strongly recommended : 1 per cent. aqueous solution of eosin with azur II, 8–9 drops of the solution is added to 50 cc. of distilled water and the slide is stained for from 30 to 45 minutes. They are unable to confirm the findings of some writers that the predominance of certain cells is characteristic of the different stages of trachoma. Polychrome erythrocytes and normoblasts were found in all stages. Lymphoblasts, some undergoing mitosis, varied in numbers from 1 to 10 per field. Lymphocytes are very plentiful in the early stage of the disease, but become more scanty with its progress and very few are found in the third stage. Eosinophiles were very rare, and this paucity

* For the twenty-fifth of this series see Vol. 33, pp. 448–453.

[1] MacCallan (A. F.). The Relationship between Conjunctivitis and Trachoma. —*Brit. Jl. Ophthalm.* 1936. June. Vol. 20. No. 6. pp. 346–350.

[2] Pulvertaft. The Nature of the So-called Follicles in the Second Stage of Trachoma (Tr. II*a*).—*Rev. Internat. du Trachome.* 1936. Jan. Vol. 13. No. 1. pp. 19–23. [10 refs.]

[3] Marcus (J. M.) & Wainer (K. G.). La cytologie de la conjonctive dans le trachome et les conjonctivites folliculaires.—*Rev. Internat. du Trachome.* 1936. Apr. Vol. 13. No. 2. pp. 68–82.

constitutes a valuable diagnostic feature. The epithelial cells show more marked degenerative changes than in other forms of conjunctivitis.

Bruck[4] has noted the peculiar rarity in Palestine of pannus in trachomatous patients, though entropion and trichiasis are common. He attributes the blindness which is rife to secondary infections by Koch-Weeks and the gonococcus. He was impressed by the early age at which trachoma develops.

Farid Massoud[5] after a study of 4,000 trachomatous patients, concluded that the disease was mainly acquired in infancy and that faulty diet and bad personal hygiene added to local irritation played an important part in its causation. Over-feeding and improper diet appeared to be more deleterious than actual under-feeding. Over-feeding is common among the babies of the poorer classes in Egypt.

Sédan[6] has recorded the history of an Armenian family in Marseilles which consisted of a trachomatous grandmother, father, mother and two elder children, with three non-trachomatous younger children. Two of the latter contracted measles and shortly afterwards developed trachoma, whilst the third remained free from both diseases. He attributes this infection to the fact that the children were looked after by their trachomatous brothers during the measles at a time when their conjunctival resistance was lowered by the disease. Their trachoma proved to be of an obstinate nature despite drastic treatment.

Charamis[7] has reported favourably upon Brecker's treatment of the disease by the injection of detoxicated bee venom beneath the conjunctiva of the lids.

Cornea.—Duggan & Nanavati[8] have reported their experience of tattooing corneal leucomata with gold and platinum chloride. Twenty-nine opacities were tattooed with gold chloride and fourteen with platinum chloride. Tannin in 1 per cent. solution, used as a reducing agent, was found to be more painful than hydrazin hydrate, and produced a lighter tint. After an interval the stained area tends to become smaller and somewhat mottled, but the final result is satisfactory.

Cataract.—Wright[9] expresses the opinion that though the question may be doubtful, cataract really is exceptionally common in India. Of 2,000 outdoor workers between the ages of 40 and 60, resident in a dry and arid district, the cataract incidence was one person in 5·1. Of 2,000 working in a humid and fertile area (West Coast) the incidence was one person in 3·4.

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[4] Bruck (A. J.). Sur le trachome en Palestine.—*Rev. Internat. du Trachome.* 1936. Apr. Vol. 13. No. 2. pp. 98–104.

[5] Massoud (Farid). Trachoma as an Endemic Disease in Egypt.—*Rev. Internat. du Trachome.* 1936. Apr. Vol. 13. No. 2. pp. 111–118. [21 refs.]

[6] Sédan (Jean). Deux cas de contamination trachomateuse familiale après conjonctivites morbilleuses préalables.—*Rev. Internat. du Trachome.* 1936. Jan. Vol. 13. No. 1. pp. 48–50.

[7] Charamis (J.). Le traitement du trachome par le virus d'abeille.—*Rev. Internat. du Trachome.* 1936. Jan. Vol. 13. No. 1. pp. 27–34.

[8] Duggan (J. N.) & Nanavati (B. P.). Tattooing of Corneal Opacity with Gold and Platinum Chloride.—*Brit. Jl. Ophthalm.* 1936. July. Vol. 20. No. 7. pp. 419–425.

[9] Wright (R. E.). Incidence of Cataract at Certain Age Periods in South Indian Districts.—*Brit. Jl. Ophthalm.* 1936. Sept. Vol. 20. No. 9. p. 545.

Glaucoma.—KIRWAN[10] has investigated the occurrence of primary glaucoma in patients suffering from epidemic dropsy. He remarks that this is the only general disease at present known in which glaucoma forms an integral part, and is consequently of vast importance as it provides such a field for research. Epidemic dropsy is an entirely different disease from beriberi ; its two outstanding manifestations are the vaso-dilatation of the whole capillary system and an increased permeability of the capillary endothelium. It appears to be associated with the ingestion of diseased, stored, parboiled rice. Gram-positive organisms with certain special characteristics are frequently found in the stools, and similar organisms have been recovered from diseased rice. The canal of Schlemm and the neighbouring tissues appear normal ; also the epithelium of the ciliary processes, but the latter are extremely hypervascular and there is great dilatation of the choroid capillaries ; inflammatory changes are, however, absent. In the aqueous humour of patients suffering from epidemic dropsy glaucoma there is a marked increase of the albumens while the globulins remain the same. In a certain number of patients suffering from the disease a histamine-like body has been proved to be present in the aqueous humour. The enormous number of 1,695 cases was seen during 1934 and 1935.

Miscellaneous.—In an article on myopia in China RASMUSSEN[11] remarks how very common the condition is in the country. He attributes this to gross over-strain of accommodation and convergence, owing to the bad conditions in which work of various kinds is carried on. Progressive myopia is, however, extremely rare. Trachoma and corneal ulcers account for most of the blindness in China.

CHAKRAVERTI[12] has furnished some excellent hints to the general practitioner on the subject of difficulties likely to be encountered in ophthalmic practice. He states " the eye is a delicate organ and as such it should be handled with extreme gentleness and care. The mildest medicines, namely, normal saline lotion and boric lotion, liquid paraffin drops and boro-vaselin should as far as possible only be prescribed." He lays proper emphasis on the connexion between inflammations of the skin and conjunctivitis, and also advocates mild applications in cases of trachoma.

BUSCHKE[13] has reviewed the rôle played by the vitamins in ocular nutrition, especially lens nutrition. He quotes the observations made by various authorities and stresses the importance of considering the mutual interaction of the different essential food factors.

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[10] KIRWAN (E. O'G.). The Aetiology of Chronic Primary Glaucoma.—*Brit. Jl. Ophthalm.* 1936. June. Vol. 20. No. 6. pp. 321–331. With 5 figs. & 1 coloured plate.

[11] RASMUSSEN (O. D.). The Incidence of Myopia in China. Data and Theses from Periodical Investigations covering Thirty Years Residence and Association with Refracting and Hospital Centres in a Score of the Larger Cities.—*Brit. Jl. Ophthalm.* 1936. June. Vol. 20. No. 6. pp. 350–360. With 1 fig.

[12] CHAKRAVERTI (C. R.). Pitfalls in Ophthalmic Practice.—*Indian Med. Gaz.* 1936. Apr. Vol. 71. No. 4. pp. 199–203.

[13] BUSCHKE (Wilhelm). Die Vitamine in der Ophthalmologie.—*Ztschr. f. Vitaminf.* Berne. 1936. Jan. Vol. 5. No. 1. pp. 37–68. [4 pages of refs.]

Toxic amblyopia is not very common in the tropics. Orr's[14] observation on its treatment with acetylcholine is nevertheless interesting. He assumes that arteriole constriction is chiefly responsible for the symptoms, and in support of his hypothesis quotes Duggan's successful use of sodium nitrite intravenously. He injects intramuscularly 0·1 gram of acetylcholine (B.D.H.) or 1 cc. of acecholin (Anglo-French Drug Co.) daily for a week, and claims very marked success in three chronic cases. He suggests that the treatment may be of use in retinitis pigmentosa.

[15]The *Bulletin of the Ophthalmological Society of Egypt* (Vol. 28, 1935) contains many interesting papers by members of the Society. Contributions to the study of conjunctival disease are, perhaps, the most important. Farid Massoud directs attention to the necessity for care as regards personal hygiene and diet of the trachomatous patient in addition to ordering local treatment. Rapid improvement in the conjunctival condition is noticed when a bath is taken daily. Most of the members agreed that infection due to prevalent ophthalmias played an important part in the incidence of trachoma. R. P. Wilson remarks that very mild cases of trachoma may be met with; these may undergo a spontaneous cure, but evidence of vascular invasion of the cornea can always be found in them. Some of the conjunctival conditions in school children diagnosed as trachoma are not trachoma at all. Ibrahim Ahmed Mahomed has found conjunctival eosinophilia to be much more common in Egyptians than he expected. Eosinophilia occurs quite often in trachomatous patients and is unconnected with spring catarrh. Wilson in a study of the seasonal incidence of acute ophthalmias was struck by the suggestive fact that the minimum summer incidence coincides with the fall in the number of flies. M. A. H. Attiah discusses the clinical aspects of phlyctenular disease in Egypt. M. Khalil describes fully Lacarrère's method of cataract extraction by diathermic coagulation. An unusual type of lipoid infiltration of the cornea is described by A. M. Soliman. M. A. Attiah and M. Sorour record their experiences of treatment of corneal ulceration by the injection of an extract of placental tissue and umbilical cord.

H. Kirkpatrick.

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[14] Orr (H. Campbell). Acetylcholine in Tobacco Amblyopia.—*Brit. Med. Jl.* 1936. July 11. pp. 69–70.

[15] Bulletin of the Ophthalmological Society of Egypt. 1935. Vol. 28. Session 32. pp. xxx+271. With numerous illustrations.

THE TYPHUS GROUP OF FEVERS.

Quarterly Bulletin of the Health Organisation. League of Nations. Geneva. 1936. Mar. Vol. 5. No. 1. pp. 140–151.—**Typhus (Rickettsiosis).** [Report of Committee on Typhus Fever and Resolutions of Pan-African Health Conference, Johannesburg, Nov. 20–30, 1935, pp. 140–141.] Cluver (E. H.). **Typhus and Typhus-like Diseases in South Africa** [Annex I. pp. 142–149]. [20 refs.] de Boer (H. S.). **Typhus Fever in Uganda and its Control** [Annex II. pp. 149–151].

i. In the opinion of the Committee tick typhus (tick bite fever) and flea-borne typhus do not constitute serious public health problems in S. Africa, they are as a rule mild diseases and are non-epidemic.

Louse typhus is, however, a very serious problem in many African States. There were approximately 1,000 deaths in the Union of South Africa during the year ending June 1935. Preventive measures being adopted consist of various methods of delousing blankets, clothing and the people themselves. It was agreed that no real progress against the spread of typhus can be made without greatly improving the economic status of the native in the States affected.

ii. The three types of typhus fever met with in South Africa, tick bite fever, rat flea typhus and louse typhus, are fully described on the lines of the work of Pijper and Dau already summarized in this *Bulletin* [1935, Vol. 32, pp. 154, 561]. Tables are given showing the incidence of louse-borne typhus in the Union of South Africa; from 1923 to 1933, 34,986 cases were reported in this period and as is pointed out probably a large number of the milder cases were never reported at all.

iii. An account of an outbreak of louse-borne typhus in the Kigezi district of Uganda.

In 1932 there were	150	cases with	10	deaths
,, 1933	,,	128	,,	20	,,
,, 1934	,,	103	,,	6	,,

These natives are clothed in goat skins with the hair left on, the skins could not be disinfected by steam as this method destroyed the skins but a method of disinfestation by means of dry heat was devised and has proved a great success.

The skins are spread over a wicker basket and dried cow dung is burned underneath, the skins could be left on the frame for many hours without damage and lice and eggs were killed in less than half an hour. [See *Bull. of Hyg.*, 1935, Vol. 10, p. 629, where an illustration of the apparatus is given.] *D. Harvey.*

Bulletin de l'Académie de Médecine de Roumanie. Paris. 1936. Vol. 1. No. 3. pp. 569–575. With 1 graph.—Rapport de la commission, nommée par l'Académie de Médecine, sur le plan de lutte contre le typhus exanthématique. [**Prevention of Exanthematic Typhus. Report of the Rumanian Academy of Medicine.**]

The first part of this report is historical and deals with the huge epidemics of typhus which devastated Serbia, Galicia and Rumania during the European War 1914–18.

Although typhus has now disappeared from Western Europe, it still smoulders on in Rumania, Russia, and Bessarabia and from these endemic centres epidemics break out every year in the winter time. The methods recommended by the Commission for combating these epidemics are similar to those evolved during the war. Diagnosis, notification, isolation and disinfestation of cases. Establishment of disinfestation stations in towns and villages and also scientific laboratories for the study of the disease. *D. H.*

PINKERTON (Henry). **Criteria for the Accurate Classification of the Rickettsial Diseases (Rickettsioses) and of their Etiological Agents.**—*Parasitology*. 1936. Mar. Vol. 28. No. 2. pp. 172–189. With 10 figs. on 2 plates. [27 refs.]

In recent years acute febrile diseases of man in nearly all parts of the world have been recognized as belonging to the rickettsia group. The occurrence of mild and atypical forms appears to be an outstanding characteristic of this group of diseases and this makes their differential diagnoses a difficult matter. The author deprecates the tendency to assign a local or personal name to organisms seen in incompletely studied strains, since this has often led to distinction without differences and to the accumulation of invalid terms in the literature. He considers that it is possible to classify accurately these diseases if complete morphological, histopathological and immunological studies are made and the object of the paper under review is to define and tabulate the criteria on which such an accurate classification may be based.

Criteria for Classification.

1. *Cross immunity tests.*—For practical purposes this is perhaps the most important single criterion, if correctly carried out and interpreted.

2. Study of smear preparations from the scrotal sac exudate of guineapigs and rats. Points to be observed are : (*a*) pattern of cell infection ; (*b*) type of cell invaded ; (*c*) morphology of organism.

In typhus, for example, the cells are packed with organisms and the serosal cells only are invaded.

In spotted fever the organisms are scanty and both serosal cells and macrophages are invaded ; the organism in spotted fever is larger than that in typhus and is more lanceolate and diplococcoid in shape and form ; a halo may also be observed.

3. *Histopathology.*—For these examinations the most suitable material for examination from the guineapig is obtained from the scrotum, scrotal sac, epididymis and testis.

" Typhus nodes " may be found in material obtained from guineapigs infected with typhus and also in those infected with mild " spotted fever " but the characteristic lesion in spotted fever is the formation of acute fibrin thrombi in arterioles and venules. These thrombi contain polymorphs and may occlude the lumen of the vessel ; the term thrombonecrosis is applied to this condition which has never been observed in animals or man infected with typhus.

4. *Location and morphology of organisms in sections.*—In typhus the mesothelial cells lining the tunica are packed with organisms but none are seen in macrophage cells nor are the smooth muscle cells invaded. In spotted fever mesothelial cells and also macrophages contain organisms but only scanty in number and the organisms are also present in the smooth muscle cells, a diagnostic point already described in 1919 by WOLBACH.

5. *Study of organism in tissue culture.*—In typhus the cells are packed with organisms but all are in the cytoplasm, none in the nucleus. In spotted fever the organisms are scanty in the plasma but the nuclei of the cell are invaded and may be packed with organisms which are mostly of the coccoid form.

In tissue culture the differences of morphology already noted in smear preparations are again noticeable in tissue cultures of the organism.

6. *Study of organism in the arthropod vector.*—In typhus the organism is found only in the cells lining the gut and is not passed hereditarily either in the louse or the flea, whereas in the tick in spotted fever all the tissues are invaded and the organism is passed hereditarily.

7. *Weil-Felix reaction.*—Useful but not decisive in differentiating mild cases of typhus from mild cases of " spotted fever."

8. *Clinical picture in the guineapig.*—The scrotal reaction in guineapigs infected with spotted fever may go on to gangrene and can be produced by subcutaneous injection of virus ; this does not occur in typhus. There is usually a high mortality in spotted fever guineapigs. Guineapigs do not die from typhus infection.

9. *Clinical picture in man.*—May be of use in severe cases but is of little use in a district where mild cases of spotted fever are occurring along with cases of endemic typhus.

The actual viruses studied by the author were as follows :—

Typhus—European epidemic strain (Wolbach)
European epidemic strain (Breinl)
Mexican endemic strain (Mooser)
North Carolina endemic strain (Maxcy)

Spotted fever—Rocky Mountain strain (Parker)
Eastern strain (Badger)
Minnesota strain (Reimann)

The paper is directly concerned with the differentiation of typhus and spotted fever although of course the criteria described would serve for the investigation and classification of any of the rickettsial diseases transmissible to laboratory animals.

The following is from the summary and conclusions of the author :—

" Morphological, histopathological, and immunological studies of seven strains of rickettsial disease have placed them in two widely separated groups, for which typhus and spotted fever seem the most logical names.

" Diseases of the spotted fever group are carried by ticks (Arachnoidea) and characterised by distinctive histopathological lesions—thrombonecrosis of arterioles and venules, with micro-organisms of definite and characteristic morphology, invading smooth muscle cells, as well as endothelium, mesothelium, and macrophages. Tissue culture studies show massive invasion and distention of cell nuclei, while cytoplasmic invasion of host cells is relatively sparse.

" Diseases of the typhus group are carried by lice and fleas (Insecta) and characterised by the facts that the intracellular parasite which causes them invades only endothelium and mesothelium, distends the cytoplasm of its host cells without ever invading nuclei, and in guinea pigs causes only proliferative endangeitis without thrombonecrosis.

" In the spotted fever tick, organisms are intranuclear as well as intracytoplasmic, invade nearly all types of tissue, and are transmitted hereditarily. In typhus-infected lice and fleas, organisms are intracytoplasmic only, infect only the lining cells of the gut, and are not transmitted hereditarily."

The author considers that " The morphological, histopathological, and immunological differences between typhus and spotted fever are so great that these two diseases, as well as their causative organisms, should be widely separated from one another, and given definitely distinguishing names.

" It is suggested that the well-established name, *Rickettsia prowazeki*, be applied to intracytoplasmic micro-organisms in Insecta causing all diseases of the typhus group, and that *Dermacentroxenus rickettsi* (the name originally given by Wolbach) be applied to hereditarily transmitted micro-organisms invading nuclei in Arachnoidea and in tissue cultures, causing all diseases of the spotted fever group. Specific varieties, when established by the demonstration of definite and constant biological differences, should be indicated by variety names, as *Rickettsia prowazeki mooseri* (for endemic flea-borne typhus in all parts of the world). *D. H.*

Boyd (J. S. K.). **Classification of Typhus Fevers.** [Correspondence.] —*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. June 30. Vol. 30. No. 1. pp. 131–133.

At the recent discussion on typhus fever at the Royal Society of Tropical Medicine references were made to a paper by Boyd [see this *Bulletin*, 1936, Vol. 33, p. 417]. As he points out in this letter the table in that paper was not intended to be a " suggested classification " but was an analysis according to serological findings of cases of typhus fever in India. This analysis allowed the cases to be placed in three sub-groups.

1. XK type=main agglutin corresponding to Japanese River fever.
2. X19 X2 type=group agglutinins corresponding to Rocky Mountain fever and boutonneuse fever.
3. X19 type=main agglutinin corresponding to endemic or murine typhus. *D. H.*

Covell (G.) & Mehta (D. R.). **Studies on Typhus in the Simla Hills. Part IV. The Rôle of the Rat Flea in the Transmission of Typhus.** —*Indian Jl. Med. Res.* 1936. Apr. Vol. 23. No. 4. pp. 921–927. With 1 chart. [15 refs.]

A hundred and twenty-one *Xenopsylla cheopis* were collected from wild rats caught in Sabathu in the neighbourhood of Kasauli where cases of typhus had recently occurred. These fleas were emulsified and injected intraperitoneally into a guineapig which developed fever and orchitis. This strain of virus has now been passaged for 17 generations; out of 50 animals inoculated 38 developed orchitis and Rickettsia were readily demonstrated in smears from the scrotum. White rats which were inoculated with emulsion of brain from infected guineapigs developed agglutinins for *Proteus OX19* in two instances up to a dilution of 1/1,000, no reaction was obtained with *OXK* or *OX2*. The reactions of laboratory animals to this strain of virus isolated from fleas were the same as were obtained with the virus previously isolated from wild rats captured in the same villages; cross immunity tests were also positive.

Forty-six *X. cheopis* fleas were fed on a white rat which had been infected with the wild rat virus, these fleas were fed for three days on a second rat but without result, but later on the survivors of the same fleas were fed for nine days on a third white rat and this animal became

infected and guineapigs were infected from it. These animals developed fever and orchitis and Rickettsia were demonstrated; also cross immunity experiments with this flea strain and the original strain were positive. Some of the fleas which were infected by feeding on this rat were taken and examined and the cells of the midgut were found to be crammed with Rickettsial bodies. No Rickettsia could be found in laboratory bred fleas. The author points out that he has now isolated a virus from the wild rats in Kasauli and also from fleas caught on wild rats; he also shows that the time of maximum prevalence of fleas coincides with the season when the greatest number of typhus cases occur.

[A point requiring explanation is why this virus isolated from rats and fleas produces agglutinins for *OX19* only, as do similar viruses elsewhere, whereas the serum of the cases agglutinates *OXK*.] *D. H.*

Mercier (H.), Fabre & Soulage. Sur un cas de fièvre typho-exanthématique d'origine vraisemblablement murine observé à Marseille. [**Case of Exanthematic Typhus, probably of Murine Origin, in Marseilles.**]—*Marseille-Méd.* 1936. Mar. 15. Vol. 73. No. 8. pp. 339–344. With 1 chart.

Many cases of boutonneuse fever have been reported in Marseilles but so far no cases of endemic typhus have been seen, although such have been reported from other Mediterranean seaports.

The patient was a soldier and was admitted to hospital from camp; the fever was severe, typhoid state, profuse rash on body, hands and feet, but sparing the face; Weil-Felix reaction positive 1/300 at beginning of second week of fever and epididymitis on the 21st day of fever. The man was a storekeeper and his work brought him into contact with rats. Experimental proof of the exact nature of the virus was not possible. *D. H.*

Olmer (D.), Olmer (J.) & Audier (M.). Les phlébites de la fièvre boutonneuse. [**Phlebitis in Boutonneuse Fever.**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 457–459.

Although the authors have seen many cases of boutonneuse fever they have so far seen phlebitis only in two cases. In one of these cases the initial sore, or tache noire, was just over the right femoral veins and the phlebitis occurred in this region. It is suggested that the phlebitis in typhus fever may be due to the Proteus organism and not to the virus itself. *D. H.*

Covell (G.). **Studies on Typhus in the Simla Hills. Part V. Attempts to establish Strains of Typhus from Human Sources.**—*Indian Jl. Med. Res.* 1936. July. Vol. 24. No. 1. pp. 139–147. [13 refs.]

In a previous paper the author has recorded the isolation of strains of typhus virus from wild rats and rat fleas in Kasauli, Punjab. The rat strain has now been passaged for 35 generations and the strain from rat fleas for 31 generations; rabbits and white rats inoculated with these strains gave a positive Weil-Felix reaction for *OX19* and negative for *OXK* and *OX2*.

The present paper deals with the isolation of typhus virus from human cases; only a few cases of typhus occurred during the year and

of these only four were admitted to hospitals. Blood from three of these cases was inoculated into guineapigs ; in all the three patients the Weil-Felix reaction was positive for *OXK* but negative for *OX19*. In the first case the blood was taken on the 11th day of the fever and in the second case on the 9th day. Two guineapigs were inoculated intraperitoneally and both developed fever, the virus was passaged for four generations and was then lost. One of the guineapigs was killed during the fever and the brain examined ; definite typhus lesions were detected around the small blood vessels. None of the guineapigs developed orchitis and Rickettsia could not be found in smears from the tunica.

In Case 3 the blood was taken on the 10th day of fever and inoculated into a guineapig ; fever developed on the 8th day and the virus was passaged for seven generations and then lost ; scrotal reaction (slight) was noted on one occasion. Emulsion of the spleen of an infected guineapig (passage virus) was inoculated into the anterior chamber of the eye of a rabbit. An iridocyclitis resulted and this virus was successfully passaged in the eyes of rabbits for four generations and then lost ; but Rickettsia could not be found in Descemet's membrane.

Monkeys inoculated with the virus showed little or no signs of illness, but one gave a positive Weil-Felix reaction for *OXK*. Of 29 rabbits inoculated 6 gave positive reactions for *OXK* and all were negative for *OX19* ; in the positive reactors there was a rising and a falling titre of agglutination.

It will be noted that there was a marked contrast between the action of these viruses obtained from the human cases and the rat virus previously isolated ; the latter caused a severe and prolonged fever in guineapigs with scrotal reaction, and numerous Rickettsia could be readily found in smears from the tunica ; also the sera of rats and rabbits yielded a marked positive reaction for *OX19*. On the other hand the virus from cases of typhus caused only slight fever in guineapigs without any scrotal reaction and Rickettsia could not be definitely identified in smear preparations ; this virus could not be established in laboratory animals and the Weil-Felix reaction was positive only in low dilutions and for *OXK*, not for *OX19*. *D. H.*

Lewthwaite (R.) & Savoor (S. R.). **Recent Work on the Typhus-like Fevers of Malaya.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. Apr. 8. Vol. 29. No. 6. pp. 561–571. [16 refs.]

This paper read before the Royal Society of Tropical Medicine and Hygiene gives an account of some of the work carried out by the authors in Malaya on the typhus group of fevers, rural typhus, tsutsugamushi fever and urban typhus ; most of this work has already been published and has been reviewed in the *Bulletin*.

The principal results obtained are grouped under five headings :— (1) Demonstration of the rickettsial nature of the viruses of all three members of the tropical typhus group in Malaya ; (2) The relationship of the three diseases to each other and to Rocky Mountain fever ; (3) Search for the carriers of rural typhus and urban typhus ; (4) Evidence as to the reservoir of the viruses ; (5) Attempted inoculation of guineapigs against experimental infection with rural typhus.

The findings are as follows :—

1. Rickettsia could be demonstrated in all three diseases ; in rural typhus and in tsutsugamushi fever they were seen in the cells of

Descemet's membrane in the eye and resembled in morphology *R. orientalis* of Japanese workers. Rickettsia seen in smear preparations from the tunica of guineapigs infected with urban typhus on the other hand resembled *R. prowazeki*.

2. Complete cross immunity exists between rural typhus and tsutsugamushi fever in Malaya ; and the viruses of these two diseases when injected intradermally in rabbits and monkeys produce a primary sore. There is no cross immunity between the viruses of urban and rural typhus or between rural typhus and Rocky Mountain fever.

3. Urban typhus in Malaya, as elsewhere, is carried by the rat flea ; but the rat flea is not the vector of rural typhus although it can be infected experimentally. Two species of tick, *Dermacentor andersoni* and *Rhipicephalus sanguineus*, were experimented with but no evidence could be obtained that either species of tick could transmit or even acquire the virus of rural or urban typhus.

4. Experiments have shown that the wild rat is a reservoir of the virus of rural typhus ; two strains of this virus were isolated from rats and when inoculated into rabbits produced agglutinins for *OXK*, and produced fever and ascites in guineapigs.

5. All attempts to protect guineapigs, by vaccination with formalized brain tissue of animals infected with the virus of rural typhus, failed.

In the discussion which followed, Dr. Felix pointed out that killed virus from the arthropod vector is superior to killed virus from mammalian tissue for immunization. Dr. Fletcher is convinced that few if any cases of typhus occurred in Malaya before 1924. Colonel Shortt referred to recent work by Covell and himself in Kasauli ; his opinion is also that typhus is a new disease in that locality. Dr. Lépine compared conditions in Indo-China with those met with in Malaya. He agreed that rural typhus and tsutsugamushi fever are probably one and the same disease. *D. H.*

Lépine (P.) & Lorando (N.). Etude d'un foyer de typhus endémique d'origine murine. [**Endemic Typhus associated with Rats in Athens.**]—*Bull. Soc. Path. Exot.* 1936. Mar. 11. Vol. 29. No. 3. pp. 285–295. With 4 figs. [10 refs.]

Endemic typhus was first reported in Athens in 1931, and since then in one small area in the city and in the same houses in that area cases have been reported every winter, whereas in other parts of the city only a few isolated cases have been discovered. This particular section of the city is a " slum area " consisting of small dilapidated houses built over the ruins of the ancient city. These sites are being gradually excavated by antiquarians and as the houses are removed the people that lived in these houses are concentrated more and more in the remaining houses and with them is concentrated the rat population ; these rats find plentiful harbourage in the underground wells, pits and cellars of the ancient historical ruins. A notable increase of rats in some of the houses was observed after the destruction of a military bakery which had been accommodated in a disused Turkish mosque.

It is undoubtedly this aggregation of people and rats which has caused the outbreaks of typhus fever each cold season. In one group of refugees living in this area in very crowded conditions in tumbledown rat infested huts no cases of typhus occurred among the adults but several cases were met with in the children. On enquiry it was

found that in Asia Minor, where these people had come from, the older people had suffered from typhus and were immune ; the children born since arrival in Athens contracted fever. The cases were typical of endemic typhus and the Weil-Felix reaction *OX19* was positive up to a dilution of 1/4,000 ; strains of murine virus were isolated by injection of the blood of the patients into the spermophile (Citellus), an animal which the authors find to be more susceptible than the guineapig, and the virus remains viable in the brain for long periods.

The strains of virus isolated from the rats and from the cases of fever were proved to be identical. Although many mice were captured in the buildings none was found to be infected. *D. H.*

Vaucel (M.). Recherche du typhus murin au Tonkin. [**Research on Murine Typhus at Tonking.**]—*Bull. Soc. Méd. Chirurg. Indochine.* 1936. Feb. Vol. 14. No. 2. pp. 251–280. With 16 charts.

——. Recherche du typhus murin au Tonkin.—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 542–550.

A hundred and thirteen rats captured in Hanoi in 1934 were examined and all were negative. In 1933, 154 rats were examined and from the brains of these animals nine viruses were isolated ; on further examination of these nine only one gave definite tests for typhus, *i.e.*, fever, scrotal reaction in guineapigs and cross immunity with a known typhus virus. A typhus virus was also obtained from the brains of rats captured in other areas in Tonking. The author points out that the local murine virus does not give so constant or definite scrotal reactions in male guineapigs as do viruses described in other countries and, owing to the slight fever produced and difficulty of establishing the virus, cross immunity test could not be satisfactorily completed. *D. H.*

Toullec (F.) & Riou (M.). Quelques cas de typhus endémique contractés au Tonkin. [**Endemic Typhus contracted in Tonking.**]—*Bull. Soc. Méd.-Chirurg. Indochine.* 1936. Feb. Vol. 14. No. 2. pp. 288–299.

Seven cases of typhus are described in detail ; in one case *Bact. typhosum* was isolated by blood culture but the Widal reaction was negative and the Weil-Felix reaction was positive up to 1/1,000. In the other six cases the Weil-Felix reaction was also positive. A rash was seen in two only of the cases and in these was mild and transient. The only constant symptom in all the cases was a very definite fever which lasted from 2 to 3 weeks. Blood was taken from one of the cases and injected into guineapigs but without result. *D. H.*

Massias (Charles). Typhus tropical bénin. Typhus endémique en Cochinchine. [**Benign Tropical Typhus in Cochin-China.**]—*Rev. Méd. et Hyg. Trop.* 1936. Jan.–Feb. Vol. 28. No. 1. pp. 38–43. [19 refs.]

This paper gives an account of cases of typhus previously described in Indo-China and Cochin-China ; in the present paper a mild case of fever is reported which occurred in the Cochin-China delta. There was no rash, no eruption and no primary sore, but the serum of the patient agglutinated *OXK* up to a dilution of 1/100. *D. H.*

MONTEL (R.). Cas de pseudo-typhus type Schüffner observés en Cochinchine, fièvre boutonneuse ? Fièvre fluviale du Japon ? [**Pseudo-Typhus of Schüffner Type in Cochin-China. Boutonneuse Fever ? Japanese River Fever ?**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 551–559.

Five cases are described.

No history of bite of tick in any ; infection was possibly due to mites. A primary ulcer was noted in 2 only out of 5 but swollen glands in all. Incubation period 5 days. Clinically the cases resembled boutonneuse fever ; but as LÉPINE pointed out in the discussion there is no cross immunity between boutonneuse fever virus and Japanese River fever virus. *D. H.*

VAN WAARDENBURG (D. A.). Over vier gevallen van Rickettsiose in de residentie Benkoelen. [**Four Cases of Rickettsia Infection in Benkoelen Residency.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. Mar. 24. Vol. 76. No. 12. pp. 712–721.

Clinical details of four patients are briefly stated and more fully the results of their blood examination and serological tests. The first was a European, 39 years old ; his serum reacted with *Proteus X19* in a dilution of 1 : 100 on the 7th day of illness, 1 : 800 on the 20th. The second was a Javanese, 30 years ; he gave agglutination of *Proteus X19* 1 : 50 on the 12th day, 1 : 800 on the 19th. The third, also a Javanese, aged about 50 years, gave a negative with *Proteus X19* but a positive 1 : 100 with the Kingsbury strain [day of illness not known for certain, he had felt ill for " several days "] and a week later 1 : 800 against the same strain. The last, a Batakian, reacted with *Proteus X19*, 1 : 100 on the 20th day and 1 : 800 on the 27th. Thus three were infected with shop-typhus and one with scrub-typhus ; it is worthy of note that two of the three were out-door workers. *H. H. S.*

TOULLEC (F.) & RIOU (M.). Le typhus murin. [**Murine Typhus.**]—*Bull. Soc. Méd.-Chirurg. Indochine.* 1936. Feb. Vol. 14. No. 2. pp. 281–287.

A general discussion of endemic typhus with reference to the virus, the carrier, the reservoir, diagnosis, clinical course in man with therapeutic treatment. *D. H.*

i. HUGHES (J. C.), DIETHELM (O. A.) & TEBBUTT (A. H.). **Australian Typhus, with a Report of a Fatal Case.**—*Med. Jl. Australia.* 1936. Mar. 7. 23rd Year. Vol. 1. No. 10. pp. 327–329.

ii. TEBBUTT (A. H.) & WARDEN (D. A.). **Australian Typhus : a Case with Recovery.**—*Ibid.* pp. 330–331. With 1 chart.

i. The first case of endemic typhus was notified in N. South Wales in 1927 ; since that year 27 cases have occurred in the northern districts of the State and in Sydney. Three cases are reported which occurred among people who worked in a rat infested warehouse ; two of these cases gave a positive Weil-Felix reaction and one case was fatal ; at post-mortem haemorrhagic consolidation of the lung (infarction ?) was noted with enlargement of the spleen. [See paper by MATHEW below].

ii. The author reports a case of fever with rigor, macular rash and drowsiness, no enlargement of spleen ; the patient was a woman living

in a good locality but the house next door had been recently pulled down and rats had invaded her house.

Blood culture was sterile and the Widal reaction negative ; on the 11th day the Weil-Felix reaction was positive in a dilution of 1/160 for *OX19* but was negative for *OXK*. *D. H.*

Mathew (R. Y.). **Notes on a Fatal Case of Endemic Typhus.**—*Health.* Canberra. 1936. Mar. Vol. 14. No. 3. pp. 24–26.

In this case the incubation period could be definitely fixed at 13 days, fever was high and a macular rash was noted, the patient developed the typhoid state and died on the 12th day. The Weil-Felix reaction, negative at first, became positive later but only in a dilution of 1/80 for *OXK*. At the post-mortem a haemorrhagic consolidation of one lung was found with enlargement of the spleen ; a similar condition has already been noted in fatal cases of Australian typhus. Three guinea-pigs were inoculated with emulsion of the brain of this case ; one of these developed fever which lasted for 8 days with loss of weight and scrotal swelling ; diplococcal bodies were found in the scrotal fluid.

D. H.

Durieux (C.). Présence à Dakar d'un virus typhique murin. [**Murine Typhus Virus in Dakar.**]—*Bull. Soc. Path. Exot.* 1936. Mar. 11. Vol. 29. No. 3. pp. 228–230.

In the port of Dakar, which is connected by rail with Casablanca where cases of typhus have been reported, rats were captured during the summer of 1935 ; 47 of these were killed and emulsions of brain in batches of 7 were inoculated into guineapigs. A virus was isolated which produced fever and scrotal swelling in all guineapigs in which it was passaged. This virus was tested against the virus previously isolated in Casablanca and complete cross immunity was demonstrated. Four cases of fever were reported and the sera of these people were tested during convalescence ; all gave a high positive Weil-Felix reaction. *D. H.*

van Slype (W.) & Bouvier (G.). Sur l'existence de la fièvre boutonneuse dans les régions de Luputa et de Port-Franqui. [**Boutonneuse Fever in the Luputa and Port-Franqui Districts of Belgian Congo.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 143–148.

In 1933 a case of suspected boutonneuse fever was reported in this district of the Belgian Congo. It was therefore decided to carry out an investigation on the following lines :—(1) inoculation of dog ticks into guineapigs ; (2) study of the Weil-Felix reaction in local dogs. Seventy-five ticks were collected, emulsified and injected into guinea-pigs with negative results. The sera of 100 dogs tested were also negative. *D. H.*

Moreira (João Affonso) & de Magalhães (Octavio). Typho exanthematico de Minas Geraes. [**Typhus in Minas Geraes.**]—*Brasil-Medico.* 1936. Aug. 22. Vol. 50. No. 34. pp. 727–731. With 5 charts.

In previous communications [see this *Bulletin*, 1935, Vol. 32, p. 157 ; 1936, Vol. 33, p. 40] the authors dealt with certain aspects of typhus in

Minas Geraes and described experimental work in connexion therewith. It seemed unlikely that all cases should be severe, that is, there was great probability that mild cases were occurring but escaped observation. Examination of suspects was therefore undertaken in three directions: (1) To test the protective power of serum of patients, whether febrile or not; (2) To test the Weil-Felix reaction; (3) To observe the results of inoculation of guineapigs with the isolated virus. The second of these has proved of high diagnostic value in Minas Geraes. Details of one series are given, those examined being members of the household of a man who had died of the disease, *viz.*, the mother, 3 brothers, and a sister of the deceased, and the husband of the last. All gave a positive Weil-Felix, 1/40–1/100, except the husband (1/20) with *OXK* and *HXK*, the other two tests giving negative results.

H. H. S.

i. Plotz (Harry) & Giroud (Paul). Culture des rickettsies du typhus exanthématique. [**Cultivation of Rickettsia of Exanthematic Typhus.**]—*C. R. Soc. Biol.* 1936. Vol. 122. No. 23. pp. 863–864.

ii. —— & ——. Culture des rickettsies de la fièvre boutonneuse. [**Cultivation of Rickettsia of Boutonneuse Fever.**]—*Ibid.* pp. 864–865.

i. The method adopted was that of Nigg and Landsteiner. Small portions of the tunica of normal guineapigs were placed in normal guineapig serum 1 part+Tyrode's solution 2 parts, 10 to 20 pieces of tissue in 3 cc. of the fluid in 25 cc. flasks. As Plotz has already shown it is necessary to exclude air from the flasks by means of rubber bungs or caps as when serum is used; if air is admitted CO_2 is lost and the medium becomes alkaline. The flasks are incubated at 37°C. for 10 days and the inoculum is small pieces of the testicle of infected guineapigs. Rickettsia were seen in films made from the cultures and guineapigs were infected by small doses of the cultures; these animals were immune when tested later. True typhus virus was subcultured 26 times and a murine virus 22 times.

ii. This virus was isolated in 1934 from a case of boutonneuse fever and has been passaged in guineapigs since then. Two strains of this virus have been cultivated by the method described in the previous paper, one has been successfully subcultured 7 times and the other 5 times. Rickettsia were seen in cells in the cultures and also in groups outside of the cells. Inoculation of 1 cc. and 0·5 cc. of the cultures produced fever in guineapigs and even 1/1,000 cc. of culture fluid produced fever, but after a prolonged incubation period. These animals were afterwards proved to be immune. Inoculation of culture into rabbits produced a positive Weil-Felix reaction. *D. H.*

Nigg (Clara). **Studies on Culture Strains of European and Murine Typhus.**—*Jl. Experim. Med.* 1936. Mar. 1. Vol. 63. No. 3. pp. 341–351.

The cultures used in these experiments were made in tunica-serum-Tyrode medium as described in previous papers by the same author. The usual inoculum was from an emulsion of the tunica of guineapigs infected with European or murine virus. The actual viruses were a European virus (Breinl) which had been cultured through 36 generations

over a period of 1½ years in the laboratory, a murine typhus virus which had been cultured through 82 generations during a period of 4 years.

It was noted that when observed over a long period the numbers of Rickettsia present in cultures of the two series were approximately equal, and when inoculated into guineapigs from time to time little or no variation in virulence was noted in either strain. It was also noted that when employing cultures of the European virus for inoculation of guineapigs scrotal reaction was more common than when brain from passage animals was employed, due probably to the greater numbers of Rickettsia in the culture material, but after the 19th generation culture the scrotal reaction was not greater than usual. The evidence of these culture experiments indicated that the murine strain of typhus virus has a definite predilection for tunica tissue and that the reaction is due to this property rather than to the numbers of Rickettsia injected; thus small doses of emulsion of brain of guineapigs infected with the murine strain produced marked scrotal reactions, whereas large doses of brain of guineapigs infected with European virus produced little or no reaction. *D. H.*

Gnutenko (M.) & Früauf (V.). La cultivation du virus du typhus exanthématique épidémique en association avec des Sarcines. [**Cultivation of the Virus of Epidemic Typhus in association with *Sarcinae.***]—*Rev. Microbiol., Epidémiol et Parasit.* 1935. Vol. 14. No. 4. [In Russian pp. 325–337. With 5 charts. [28 refs.] French summary pp. 337–338.]

Emulsion of brain of a guineapig infected with typhus virus was inoculated into tubes of broth containing yeast cells and these tubes were incubated; a Gram-negative bacillus was isolated from one of these cultures morphologically resembling a *proteus* bacillus. This bacillus was agglutinated by the serum of typhus cases to the same degree as was the laboratory strain of *Proteus X19*. The serum of rabbits, which had been inoculated with this bacillus, agglutinated the homologous bacillus and the laboratory strains of *Proteus* to the same dilutions. *D. H.*

Baltazard (M.). Multiplication des virus exanthématiques dans les tissus. [**Multiplication of Typhus Virus in the Tissues.**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 403–411. With 2 figs.

Boutonneuse fever.—Intradermal injection of the virus of boutonneuse fever in guineapigs produces an inflammatory reaction and definite multiplication of the virus at the site of injection; the virus is present in the lesion from the 2nd to the 7th day. Histological examination shows that at first lymphocytes, plasmocytes and polynuclear cells are present but later on definite nodules consisting of groups of histiocytes can be seen around the blood vessels and Rickettsia can be seen in the cells; later still necrosis occurs.

Virus of murine typhus.—Similar results are obtained with this virus; a "tache noire" is produced by intradermal injection. The virus of Rocky Mountain fever did not produce any local reaction nor could the virus be recovered from the site of injection; the same is true of the virus of true epidemic typhus. *D. H.*

i. Brigham (George D.). **A Strain of Endemic Typhus Fever isolated from the Brain of a Wild Rat.**—*Public Health Rep.* 1936. Mar. 27. Vol. 51. No. 13. pp. 337–339.

ii. ——. **Susceptibility of the Opossum** (*Didelphis virginiana*) **to the Virus of Endemic Typhus Fever.**—*Ibid.* pp. 333–337. With 4 figs.

i. A case of typhus was discovered in a small grocery store in Alabama which was rat-infested. Rats (*R. norvegicus*) were captured and killed and fleas (*X. cheopis*) removed. The fleas were emulsified and injected into guineapigs without any result. Emulsion of the brains of the rats was injected into guineapigs and fever resulted with swelling of the scrotum. This virus was passaged for 3 generations and was also injected into rabbits; the Weil-Felix reaction became positive. Rickettsia were found in smears from the tunica of guineapigs, and typical typhus lesions were demonstrated in the brain.

Cross immunity tests were positive with a local strain of endemic typhus virus and with a strain of European epidemic typhus (Breinl).

ii. A strain of the virus of endemic typhus obtained from local cases was utilized and an opossum was inoculated intraperitoneally with testicular washings from an infected guineapig. There was no fever and no symptoms and the animal was killed on the 10th day after the inoculation (no p.m. sign); emulsion of the brain was injected into guineapigs and fever and scrotal reaction resulted. Cross immunity tests with this virus and the local virus were positive, and the brains of infected guineapigs showed typical typhus lesions. The serum of rabbits inoculated with the opossum virus gave a positive Weil-Felix reaction. *D. H.*

Masayama (Suguru). Experimentelle Untersuchung ueber die Ansteckung von Flecktyphus durch die Nasenhoehle und den Verbreitungsweg desselben im Koerperinneren. [**Experimental Research on Infection with Typhus through the Nasal Cavity and its Mode of Spread to Internal Organs.**]—*Jl. Oriental Med.* 1936. June. Vol. 24. No. 6. [In Japanese pp. 1193–1216. With 1 plate, 3 charts & 1 fig. [15 refs.] German summary pp. 87–89.]

The author finds that the virus of typhus can pass through the normal mucous membrane of the nose in guineapigs; investigation of the infected animals showed that the virus could be recovered from the lymph glands draining this area and from other glands and also from the brain and spleen. The presence of the virus in these organs was demonstrated by inoculation of emulsions into the testicle of normal guineapigs. *D. H.*

Blanc (Georges) & Baltazard (Marcel). Longévité du virus du typhus murin chez la puce (*Xenopsylla cheopis*). [**Longevity of the Murine Typhus Virus in the Flea** (*X. cheopis*).]—*C. R. Acad. Sci.* 1936. Apr. 27. Vol. 202. No. 17. pp. 1461–1463.

Laboratory bred fleas (*X. cheopis*) were fed on infected rats, then removed and placed in glass bottles; clean rats were placed in the bottles

and left for 48 hours and then removed and the fleas were returned to the bottle, the fleas were changed to a clean bottle every 10 days. There were 48 hours between the feeds on rats. The rats were killed from time to time and emulsions of brain injected into guineapigs ; also emulsion of the fleas was injected at intervals. It was found that fleas were still infective up to 100 days. The experiment started on 23rd December with 635 fleas and finished on 30th May with 35 fleas.

D. H.

Blanc (Georges) & Baltazard (Marcel). L'influence du jeûne sur le développement du virus du typhus murin chez la puce (*Xenopsylla cheopis*). [**Influence of Fasting on the Development of the Murine Typhus Virus in the Flea** (*X. cheopis*).]—*C. R. Acad. Sci.* 1936. June 29. Vol. 202. No. 26. pp. 2191–2192.

Fleas were fed on an infected rat for 24 hours, then starved for 12 days. They were then found to be non-infective ; the fleas were again fed for 48 hours and became infective.

The authors consider that the ingested virus does not multiply until a second blood feed. [A similar phenomenon has been noted with ticks and Rocky Mountain fever virus.] *D. H.*

i. Epstein (G. W.) & Silvers (I. L.). **Transmission of Rat Virus to Guinea Pigs through Fleas. Second Preliminary Report.**—*Giorn. di Batteriol. e Immunol.* 1935. May. Vol. 14. No. 5. pp. 1079–1088. With 1 fig.

ii. ——, —— & Exemplarskaya (E. V.). **Searching for Typhus Virus in Moscow Rats (Winter 1934). Third Report.**—*Ibid.* pp. 1089–1098.

i. Three hundred and seventy-one wild rats were captured in Moscow and 1,375 fleas collected from them, 3·7 fleas per rat. 1,081 fleas were utilized in the experiments and 82 per cent. of these were *X. cheopis*, 18 per cent. *Cer. fasciatus*. 412 fleas were fed on guineapigs and 245 were utilized to provide suspension of intestine for inoculation into 19 guineapigs ; in some instances the fleas were first fed on the guineapig, then killed and the intestines removed and emulsified and inoculated. In 11 groups both methods were successful. Emulsions of the intestines of 10 to 30 fleas were utilized and groups of 8 to 35 fleas were fed on the guineapigs. In one instance an emulsion of only 10 intestines produced infection and 53 per cent. of the experiments gave positive results. All these successful results were obtained with *X. cheopis* ; no success with *C. fasciatus*.

The rats from which the fleas were taken gave a positive Weil-Felix reaction and all the infected guineapigs had fever and scrotal swelling ; Rickettsia were demonstrated in smears from the intestines of the fleas and from the tunica of infected guineapigs. One conclusion was that the virus is more concentrated in the flea intestine than in the rat brain.

ii. In the winter of 1934 the same experiments were repeated but without any success, many of the experimental animals died of pneumonia but there was no evidence of typhus infection. 237 rats and 842 fleas were employed. *D. H.*

i. Balteanu (I.) & Constantinesco (N.). Réaction de Weil-Felix chez les rats sauvages de Roumanie. Présence d'un virus murin. [**Weil-Felix Reaction in Wild Rats of Rumania. Presence of a Murine Virus.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 13. pp. 1427–1430.

ii. —— & ——. Virus murin isolé à Jassy. Réceptivité des animaux de laboratoire. Sensibilité de l'homme. [**Murine Virus of Jassy. Receptivity of Laboratory Animals ; Sensitivity of Man.**]—*Ibid.* pp. 1430–1433.

iii. —— & ——. Virus murin isolé à Jassy. Dispersion et persistance dans l'organisme ; voies d'élimination et voies d'infection. [**Murine Virus of Jassy. Dispersion and Persistence in the Organism, Routes of Elimination and of Infection.**]—*Ibid.* pp. 1434–1436.

iv. —— & ——. Sur un virus murin de fièvre exanthématique isolé à Jassy (Roumanie). [**Murine Typhus Virus isolated at Jassy (Rumania).**]—*Bull. Acad. Méd. Roumanie.* Paris. 1936. Vol. 1. No. 3. pp. 518–539. With 5 figs.

i. One hundred and twenty-one rats were captured in the town of Jassy in Rumania and in villages in the neighbourhood where cases of endemic typhus had occurred. The blood of 104 of these rats was tested for the Weil-Felix reaction against emulsions of *OX19* and grouped according to the results.

Group	I	7 rats	1/200 to 1/600
,,	II	38 ,,	1/50 ,, 1/100
,,	III	59 ,,	negative

43 per cent. of the rats examined gave a positive result.

The rats were killed and the brains emulsified and injected into guineapigs. Of the 121 examined 110 gave negative results.

Four strains of virus were isolated from groups of rats, only one of these strains was established in guineapigs, this particular strain, called " Dana," was isolated from a group of 4 rats captured in Jassy at a flour mill of that name. These rats were from Group I above in which the Weil-Felix reaction was positive in 1/200 to 1/600.

ii. In this paper the reactions of the strain of typhus virus in experimental animals are recorded.

In white rats this strain produced high fever and many of the infected animals died. In wild rats fever was produced and loss of weight occurred. In mice the strain produced a fatal illness—all the infected animals died. Both in white rats and wild rats and also in the dog the Weil-Felix reaction became positive. In the spermophile only an inapparent infection resulted but the brain was infective for guineapigs for long periods.

The virus was passaged in guineapigs for many generations; emulsion made of the brains of these animals in the first few generations when inoculated into men produced typical attacks of mild typhus but when the infective material was taken from guineapigs later in the series no fever was produced but the blood of the patient was infective for guineapigs. Material taken still later in the series produced neither fever nor any infection ; the Weil-Felix reaction was positive in the human cases of fever.

iii. In guineapigs this strain of virus produced fever only, without any scrotal reaction, nor could Rickettsia be discovered in smears from

the tunica. The incubation period was usually about 3 days and fever lasted from 6 to 11 days ; there was loss of weight and 50 per cent. of the animals died. Those that recovered from the infection were immune to further inoculations with the same virus, but so far as one can discover no cross immunity tests with known strains of typhus virus were attempted.

iv. The information contained in this is the same as that given in the three preceding papers. *D. H.*

Balteanu (I.) & Constantinesco (N.). Sur la nature du virus murin de fièvre exanthématique isolé à Jassy. [**Nature of the Murine Typhus Virus isolated at Jassy.**]—*C. R. Soc. Biol.* 1936. Vol. 122. No. 21. pp. 702–704.

The reactions of this murine virus as regards its action on animals have already been described.

It was found that the virus could resist cold for 50 days and that up to 5 days bile had no effect on it. In 50 per cent. glycerine the virus was viable for 25 days but not after 60 days.

Filtrability.—Five times out of 9 trials the virus passed through a Seitz filter ; it was partially retained by a Chamberlain L2 filter and completely retained by L3. *D. H.*

Blanc (Georges) & Noury (M.). Sur un microbe du type *Proteus X19* isolé du sang d'un malade atteint de typhus murin. [**Bacillus of *Proteus X19* Type isolated from Blood of Man infected with Murine Typhus.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 15. pp. 1572–1573.

A freely mobile non-Gram-staining bacillus was isolated from the blood of a case of endemic typhus. This bacillus did not ferment lactose or liquefy gelatine but produced acid in glucose and saccharose, and acid and gas in maltose, the indole test was positive. Both H and O varieties were isolated. The O type was agglutinated up to a dilution of 1/6,000 by the serum of the case of fever. *D. H.*

Costanzi (Carlo). Note istologiche intorno a tre casi di " febbre eruttiva." —*Policlinico.* Sez. Med. 1936. Apr. 1. Vol. 43. No. 4. pp. 175–204. With 11 figs. [38 refs.]

Lewthwaite (R.) & Savoor (S. R.). **The Typhus Group of Diseases in Malaya. Part IV : the Isolation of Two Strains of Tropical Typhus from Wild Rats.**—*Brit. Jl. Experim. Path.* 1936. June. Vol. 17. No. 3. pp. 208–214.

It has already been shown by the authors and others that agglutinins for *OXK* and for *OX19* can be found in the sera of wild rats in Malaya.

The authors have succeeded in isolating a rural typhus virus from the local wild rat ; this virus produced fever and scrotal swelling in guineapigs but only agglutininis for *OXK* in rabbits. The virus of rural typhus does not as a rule produce scrotal swelling in guineapigs and in this instance the authors consider that the symptom was caused by

secondary infection with the spirillum of rat-bite fever. The typical ocular reaction was obtained in rabbits, and rickettsia resembling *R. orientalis* could readily be demonstrated in the cells of Descemet's membrane. Of 42 rabbits inoculated with this virus (Rat 5) 27 gave a positive reaction, 18 of these gave a reaction with *OXK* and 8 with *OX19*, 1 gave reactions for both. Cross immunity was complete in rabbits against the viruses of rural typhus and tsutsugamushi fever derived from human cases. *D. H.*

LEWTHWAITE (R.) & SAVOOR (S. R.). **The Typhus Group of Diseases in Malaya. Part V : The Weil-Felix Reaction in Laboratory Animals.** —*Brit. Jl. Experim. Path.* 1936. June. Vol. 17. No. 3. pp. 214–228. [12 refs.]

This highly technical paper is a report of the inoculation of rabbits and monkeys with the viruses of rural and urban typhus and the results of the Weil-Felix reaction. Both living and alcohol treated emulsions of *Proteus* were employed ; the sera of 310 normal animals were first tested with the following results :—

With *OXK* 90 per cent. of the normal sera gave negative results and with *OX19* 99·4 ; as a result of these tests it was decided that with *OXK* a titre of 50 or over would be taken as positive whereas with *OX19* a titre of 1/25 or over would be taken as positive. Blood was drawn from the test animals before inoculation and at intervals of 5 days from the 10th day onwards.

Rural typhus virus.—Of 27 rabbits inoculated intraperitoneally 18 gave positive results and all with *OXK*. Two rabbits inoculated intraocularly with the blood from two cases of rural typhus both gave positive Weil-Felix reactions with *OXK*. Of 24 rabbits inoculated intraocularly with passage virus 10 were positive all with *OXK*.

Virus of tsutsugamushi fever.—One rabbit inoculated intraperitoneally gave a positive (*OXK*). Of 6 rabbits inoculated intraocularly with the virus from cases of fever two gave a positive reaction, and of 57 inoculated by the same route with passage virus 34 gave a positive reaction with *OXK*.

Virus of urban typhus.—Of 8 rabbits inoculated intraperitoneally 4 gave a positive reaction with *OX19* and of 55 inoculated intraocularly 30 gave a positive reaction all for *OX19*.

Two strains of rural typhus virus isolated from wild rats were also tested ; these gave positive reactions in rabbits for *OXK* in the same proportion of the animals whatever route was employed. It was noted also that small doses of virus, say 0·1 cc., when inoculated into the eye of rabbits produced a much higher response than did larger doses, 6–10 cc., inoculated intraperitoneally. It was also noted that the ocular reaction in rabbits may be positive, and often is, though the Weil-Felix reaction is negative. Only 50 per cent. of rabbits inoculated give a positive with rural typhus virus, whereas with louse-borne typhus virus practically all give a positive reaction.

Monkeys—rural typhus virus.—One gibbon inoculated with the virus of rural typhus gave a positive reaction for *OXK*, 1/170, on the 21st day and a positive reaction for *OX19* on the 15th day ; this phenomenon is discussed later. Three *Macacus* monkeys all gave a positive reaction for *OXK* and negative for *OX19*.

Change of type of antibody response.—In one case of rural typhus fever the serum of the patient gave a positive reaction in rising and

falling titre both for *OX19* and for *OXK*, the *OX19* agglutinins appearing earlier ; on the 11th day of the fever the actual reasult was *OXK* 1/1,100 and *OX19* was 1/1,900 ; this virus when isolated in guineapigs gave the definite reactions of a true rural typhus virus, *i.e.*, no scrotal reaction, ascites and a 90 per cent. mortality in the animals and also gave the typical ocular reaction in rabbits with *R. orientalis* present in the cells. When inoculated into rabbits all reacted with *OXK*, but when inoculated into a gibbon the serum of this animal gave a positive reaction for both *OX19* and *OXK*, just as the serum of the original patient had done. This strain from the gibbon when inoculated into rabbits gave at first slight reactions for *OX19* and pronounced agglutinins for *OXK*, but later passages in rabbits only gave agglutinins for *OXK*.

There was definite cross immunity between this strain derived through the gibbon and the original human strain in guineapigs ; another gibbon immune to rural typhus gave no response when inoculated with this strain. Three monkeys inoculated gave agglutinins for *OXK* and none for *OX19*. *D. H.*

Pergher (G.). L'importance de la réaction de Weil-Félix dans la prophylaxie du typhus exanthématique. [**Value of the Weil-Felix Reaction in Typhus Prophylaxis.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 89–104. [26 refs.]

This paper is written in connexion with the recent epidemic of louse-borne typhus in the district of Urundi in the Belgian Congo.

It was found that it was not possible to diagnose cases of typhus from other fevers in natives on clinical grounds alone partly owing to the difficulty experienced in examining the natives and for other reasons ; for a diagnosis of typhus (which was essential before effective prophylaxis could be applied) it was necessary to rely on the results of the Weil-Felix reaction. A discussion on the nature of the Weil-Felix reaction and the relation between *Proteus* and Rickettsia follows ; and the fact that the Weil-Felix reaction is not positive in other diseases is emphasized. The sera of over 2,000 local natives, some of whom were healthy and some suffering from fevers, sleeping sickness, relapsing fever, leprosy, syphilis, etc., were tested for the Weil-Felix reaction and all were negative. This opinion has to be modified, for later in the paper it is stated that 13 per cent. of the 2,658 sera tested gave a positive reaction in a dilution of 1/160, therefore a titre of 1/320 was taken as positive. This was possible because the actual cases of typhus in Urundi all gave positive reactions in high dilutions even up to 1/50,000. Sixty per cent. of the cases gave a reaction in a dilution of 1/5,000. Some of the cases of typhus gave positive reactions for many months after the fever had ceased ; it was noted that this depended not so much on the severity of the fever but occurred in the cases from certain districts. For agglutination tests fresh emulsions of *Proteus* bacilli were utilized and a reading was made after 1 hour in the incubator, a second reading 4 to 6 hours later and a third at 12 to 18 hours. In positive cases the titre was found to be doubled at the second reading.

The difficulties of diagnosis were overcome by close collaboration between clinic and laboratory and prophylaxis based on this diagnosis was a complete success. *D. H.*

Pasricha (C. L.), Bannerjee (K.) & Lal (S.). **Immunological Methods in the Determination of Infection in a Random Sample of Hospital Admissions. Part II. (The Frequency and Concentration of Agglutinins for *Proteus X* Strains in a Series of Hospital Patients.)**—*Indian Med. Gaz.* 1936. June. Vol. 71. No. 6. pp. 320–322.

The sera of 280 patients in hospital were tested against O emulsions of *X19*, *X2* and *XK*. These people were all natives of India and were suffering from various diseases but none had typhus and none gave a history of recent typhus infection.

The results were as follows :—

	Number tested	1/25	1/50	1/100
		Per cent.	Per cent.	Per cent.
X19	280	55·7	23·2	1·4
X2	259	11·2	2·7	—
XK	77	68·8	29·8	7·8

D. H.

Blanc (Georges), Noury (M.) & Baltazard (M.). L'état de prémunition contre le typhus exanthématique chez l'homme. Simple et double vaccination. [**Premunition in Exanthematous Typhus in Man : Single and Double Vaccination.**]—*Bull. Acad. Méd.* 1936. July 7. 100th Year. 3rd Ser. Vol. 116. No. 26. pp. 33–37. With 3 charts.

Thirty-four persons were inoculated with a bile treated virus ; there was no reaction in any of them and in none was there a positive Weil-Felix reaction ; the same virus, untreated, produced a typical mild attack of fever. Eight of these inoculated people were given a test dose of virus on the 12th day after inoculation ; there was no reaction whatever ; after a further 27 days they received another test dose and again did not react.

The authors consider that up to the 12th day protection is due to the presence of the virus in the body (premunition) and after the 12th day when the virus has disappeared protection is due to immunity produced by the vaccine. *D. H.*

Blanc (Georges) & Noury (M.). Le phénomène de prémunition chez le cobaye au cours de l'infection typhique. [**Phenomenon of Premunition in the Guineapig in Course of Typhus.**]—*C. R. Soc. Biol.* 1936. Vol. 122. No. 20. pp. 522–523.

If guineapigs are inoculated with the virus of true epidemic typhus and on the 1st and 2nd day after inoculation are reinoculated with the virus of murine typhus, they develop fever and scrotal swelling ; but if they are reinoculated from the 3rd to 7th day fever follows but no scrotal swelling is seen and the fever is due to the true typhus virus only. If these animals which have been reinoculated during the incubation period are killed during the period of fever, only the virus of true typhus can be isolated from them. *D. H.*

Donatien (A.) & Lestoquard (F.). Existence de la prémunition dans la Rickettsiose naturelle ou expérimentale du chien. [**Premunition in Rickettsiosis of Dogs.**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 378–383.

The authors have already reported this Rickettsiosis of dogs which is carried by the dog tick *R. sanguineus*. The acute illness is severe and the disease is often fatal; Rickettsia can be readily demonstrated in the lungs and liver of sick animals by puncture during life, and in the brain post-mortem. The virus can be demonstrated in the animals for some months after the fever has ceased. *D. H.*

Ciuca (M.), Balteanu (I.) & Constantinesco (N.). Réceptivité du chat au virus exanthématique. Infection spontanée. Forme inapparente. [**Susceptibility of the Cat to Typhus Virus: Spontaneous Inapparent Infection.**]—*Bull. Acad. Méd. Roumanie.* Paris. 1936. Vol. 1. No. 2. pp. 271–274.

——, —— & ——. Contribution à l'étude épidémiologique du typhus. Réceptivité du chat au virus exanthématique. Infection spontanée inapparente.—*Arch. Roumaines Path. Expérim. et Microbiol.* Paris. 1936. June. Vol. 9. No. 2. pp. 283–297. With 14 figs.

A normal cat was fed on the tissues of an infected guineapig; the cat did not develop fever and the Weil-Felix reaction was negative; the animal was killed on the 37th day of the experiment and emulsion of the brain injected into guineapigs. Typical fever developed and the animals were later shown to be immune to typhus virus. A second and third cat were treated in the same way and both became infected, although neither had any fever or other symptoms of illness. Half of the brain of an infected guineapig was injected intraperitoneally into a cat, but no fever followed although later the brain of the cat was proved to be infective.

Three cats were taken from houses where cases of typhus had occurred; these animals were killed and the brain emulsion inoculated into guineapigs and they developed fever. Three cats were procured from a district where no typhus cases had occurred and these animals proved to be non-infective. *D. H.*

Blanc (Georges). Histoire et enseignements d'une vaccination contre le typhus exanthématique faite à Petitjean (Maroc). [**Typhus Vaccination at Petitjean (Morocco).**]—*Rev. d'Hyg. et de Méd. Préventive.* 1936. Apr. Vol. 58. No. 4. pp. 252–272. With 10 figs.

Up to the present no fewer than 11,216 persons have been vaccinated with living bile-treated typhus virus by the author and his co-workers. In this paper details are given of the vaccination of the entire population of a town of 6,000 inhabitants in one day. At the time when these vaccinations were carried out cases of typhus were occurring in the town and in villages near at hand; no attempt at decontamination of the people or their clothing was made.

The method of the preparation of this vaccine has already been described but very full instructions are given in the present paper;

roughly the method is that infected guineapigs are killed on the spot and emulsions are made from the tunica, the spleen and suprarenal glands ; ox bile to a strength of 5 per cent. is added to the emulsion and allowed to act for exactly 15 minutes, then 1 cc. of the bile treated vaccine is inoculated under the skin. One guineapig supplies sufficient vaccine to inoculate about 1,000 persons.

The organization on this occasion was so complete that 8,234 persons were vaccinated in one day at a rate of about 30 per minute. Three people developed fever within 24 hours ; 12 developed slight fever 10 days later, these fevers were probably due to the vaccine, which is of course a living virus ; the strain is a murine one which produces only a very mild fever in man. Nine cases of typhus occurred among the vaccinated ; these were cases of true louse-borne typhus and not murine typhus. Twenty-seven days after the vaccination there were no more cases of typhus among the vaccinated although cases continued to occur for some months among the people in the surrounding country who had not been inoculated. Although so many were inoculated with a living virus these people in no way formed a source of infection for others as the ectoparasites such as fleas and lice are unable to obtain the virus from the blood of those inoculated and these people rapidly acquire an immunity. *D. H.*

Gaud (M.). Sur les résultats de la vaccination contre le typhus exanthématique par la méthode de Georges Blanc (épidémie de Petitjean). —*Bull. Office Internat. d'Hyg. Publique.* 1936. Apr. Vol. 28. No. 4. pp. 664–667.

Giroud (Paul). Comportement du virus du typhus historique de Tunis au cours de la carence et de la précarence C. [**Behaviour of the Historic Typhus Virus (Tunis) in relation to Normal and Deficient Diets.**]—*C. R. Soc. Biol.* 1936. Vol. 121. No. 8. pp. 714–715.

Guineapigs were fed on a diet which had been deprived of all vitamin C. These animals were then inoculated with the virus of epidemic typhus, the majority of the animals developed an orchitis and severe fever but when the strain was passaged to animals on normal diet no abnormal symptoms were noted. *D. H.*

Giroud (P.). Action de la carence C sur l'immunité acquise des cobayes infectés de typhus exanthématique. [**Influence of Diet on Acquired Immunity to Typhus in Guineapigs.**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 372–373.

The authors took 30 guineapigs which had been previously inoculated with typhus virus and had recovered. These were divided into two groups ; one group was fed on an ordinary diet and the other group on a diet deficient in vitamin C. All the animals were then given a test dose of a typhus virus ; none reacted, but from two of the animals fed on C deficient diet the virus was recovered from the brain. The author argues that deficiency of C may give rise to latent infections even in protected animals and that this may in part explain the association of typhus epidemics with famine. *D. H.*

Kawamura (Kazuo). **Verification of Virus (Rickettsia) in the Urine of Patients and Guinea Pigs infected with Typhus Fever in a Broad Sense.**—*Jl. Oriental Med.* 1936. May. Vol. 24. No. 5. [In Japanese pp. 1051–1063. With 1 plate. English summary. p. 75.]

The urine of patients suffering from typhus was injected intraperitoneally into guineapigs and these animals became infected if the urine was taken in the first week of the fever. The urine of infected guineapigs was also proved to be infective for other animals when injected intraperitoneally. *D. H.*

Le Chuiton (F.), Berge (Ch.) & Pennanéac'h (J.). Inoculation au cobaye par la voie testiculaire de sang de malades atteints du typhus endémique bénin de Toulon. Recherche de l'immunité des souches isolées vis-à-vis d'une souche murine. [**Testicular Injection into Guineapigs of Blood from Individuals with Benign Endemic Typhus of Toulon. Immunity Tests with Viruses isolated *vis-a-vis* a Murine Strain.**]—*Bull. Soc. Path. Exot.* 1936. June. 10. Vol. 29. No. 6. pp. 632–638.

The blood of 16 cases of typhus from cruisers in Toulon Harbour was inoculated into the testicle of guineapigs. The clinical diagnosis was established in all the cases and the Weil-Felix reaction was positive. 3 cc. of the blood was inoculated intraperitoneally and 1·5 cc. into the testicle ; the guineapigs were observed for 24 days and fever usually commenced from the 8th to the 13th day. By the intratesticular route, 11 times a strong positive reaction, 4 times a mild reaction, 1 negative. By the intraperitoneal route, 2 gave a strong positive, 5 gave a mild reaction, 2 of the guineapigs died, 7 were negative.

D. H.

Zia (Samuel H.) & Wu (Chao Jen). **Serum Treatment of Experimental Typhus.**—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 270–274. With 4 charts.

The authors obtained a supply of anti-typhus horse serum from Professor Zinsser. Doses of 1 cc. of this serum were given to guineapigs at intervals of 48 hours after infection with the local (Peiping) strain of typhus virus ; there was definite evidence of protection. When a single dose of 2 cc. of serum was given the results were still better. Even when the fever had commenced before the dose of serum was given there was some evidence of protective power—a milder infection with shorter duration of the fever. *D. H.*

Flippin (Harrison F.). **Rocky Mountain Spotted Fever in Pennsylvania.**—*Amer. Jl. Med. Sci.* 1936. May. Vol. 191. No. 5. pp. 685–689. With 1 fig. & 1 chart.

The author describes a typical case of Rocky Mountain fever, eastern type, which occurred in Pennsylvania ; the patient was a woman aged 45, who two weeks before the fever developed had removed and crushed the ticks from her pet dog. A profuse general rash which involved also the palms of the hands and soles of the feet is illustrated. [The

excellent photos illustrating this point remind one of those already published of cases of boutonneuse fever.] The Weil-Felix reaction was positive for *X19* and *X2* in a dilution of 1/200, negative for *XK*.

D. H.

Maxcy (Kenneth F.). **The Weil-Felix Reaction of the Rabbit in the Diagnosis of Rocky Mountain Spotted Fever (Eastern Type).**—*Jl. Infect. Dis.* 1936. May–June. Vol. 58. No. 3. pp. 288–292. [10 refs.]

The Weil-Felix reaction is usually relied on for the diagnosis of Rocky Mountain fever ; but for various reasons it may not be possible to obtain serum—for instance in fatal cases the agglutinins may not have had time to develop. Maxcy therefore has employed the method of inoculating the blood of cases of fever into rabbits and testing the Weil-Felix reaction of the serum of the rabbits later. Eight cases have been tested in this way. Emulsions of formolized non-motile *X19* and *X2*, pure *O* varieties, are employed and the macroscopic method is used. The emulsion and dilution of serum are kept for 4 hours at 55° and then in the refrigerator overnight at 4°C. In order to obtain a positive reaction it is essential that the blood should be taken from the patients before the 9th day of the fever. 3 cc. of citrated blood is injected intraperitoneally into the rabbit within one hour of withdrawal from the patient. For a positive reaction to be recorded it is necessary that there should be rising and falling titre of agglutination in the rabbit serum. Of the 8 cases 2 were fatal ; 6 of the 8 rabbits developed agglutinins, 4 for *X19* and 2 for *X2*. The agglutinins rapidly disappeared from the blood of the rabbits. The two failures are accounted for by the fact that in one instance the case was very mild and the blood was not taken until the 9th day ; in the other case the blood, owing to a mistake, was kept for 4 hours after withdrawal before inoculation.

D. H.

Kuroda (Osamu). Experimentelle Studien ueber eine intrakutane Reaktion bei Tsutsugamushikrankheit. [**Experimental Study of an Intracutaneous Reaction in Tsutsugamushi Disease.**]—*Zent. f. Bakt.* I. Abt. Orig. 1936. Mar. 24. Vol. 136. No. 3/4. pp. 163–172. [23 refs.]

The author refers to earlier work in which emulsions of infected lice were used as antigen for intradermal injection for diagnosis of true typhus cases ; later on emulsions and filtrates of *Proteus X19* have been employed for the same purpose.

The author has utilized filtrates of *OXK* for intradermal injection for diagnosis and prognosis in tsutsugamushi disease. He considers that *XK* is not merely a variant or mutant of *X19* but is a distinct individual germ differing morphologically, biologically and serologically from *X19*.

These filtrates were injected intradermally into healthy young men and in 100 per cent. of cases a positive reaction resulted after an incubation period of one or two hours—occasionally in children and old men a negative result is obtained.

The test was employed also in people suffering from fevers, typhoid, tuberculosis, etc., and all gave a positive result.

In tsutsugamushi fever in the first few days a positive reaction was obtained, but after the 6th or 7th day a negative reaction resulted and this negative condition persisted for at least a month, and often longer ; in very severe cases the positive reaction could be obtained till later in the fever ; it is on this point that the author relies for aid in prognosis. Various experiments were carried out on rabbits and guineapigs and with the antisera prepared from rabbits or obtained from convalescents the results of these experiments show that the reaction is a true allergic reaction. *D. H.*

DENGUE AND SANDFLY FEVER.

Hanson (Henry). **An Epidemic of Dengue.**—*Amer. Jl. Public Health.* 1936. Mar. Vol. 26. No. 3. pp. 256–258.

This paper contains an account of an epidemic of dengue in Florida which was controlled by an active campaign directed against the breeding places of *Aëdes aegypti*. In contrasting the measures required when dealing with prevention of yellow fever it is pointed out that in yellow fever there is a solid and lasting immunity, whereas after dengue immunity is only partial and may only last for a few weeks.

If dengue breaks out in a community in which dengue has not occurred for some years it is necessary not only to reduce the number of mosquitoes but to remove them entirely, whereas in yellow fever in a community where immune people are present reduction of numbers of mosquitoes may be sufficient to check the disease ; entire elimination is not essential.

In Miami the city was divided into 50 districts and an inspector and staff were appointed for each district.

The author emphasizes the necessity of providing trained workers for supervision ; inspection of areas in which relief workers had been employed revealed numerous breeding places which had been overlooked.

The " container born " mosquitoes must be reduced to zero to check dengue ; even a more intense campaign than is required to stop yellow fever. *D. Harvey.*

Hanson (Henry). **Some Observations on Dengue.**—*Amer. Jl. Trop. Med.* 1936. May. Vol. 16. No. 3. pp. 371–375.

This is an account of the prevention and stamping out of dengue by means of antilarval methods ; a previous paper by the same author is summarized above.

A favourite site for breeding *A. aegypti* is in old motor tyre covers ; 112 tyres in one heap were all found to be breeding places.

As already pointed out by the author it is necessary to reduce the number of mosquitoes to zero in order to stamp out dengue, whereas in the case of yellow fever reduction of numbers may be sufficient.

D. H.

Sofiev (M. S.). [**The Blood-feeding Habits of Sandflies.**]—*Bull. Inst. Epidemiol. & Microbiol.* Tashkent. 1935. Vol. 3. No. 1. pp. 52–55. [In Russian.]

Using the sera of man, horse, donkey, sheep, ox, dog, rat and frog, the author employed the precipitin reaction to determine the nature of

the blood ingested by sandflies caught in Tashkent. The results of the examination of 496 sandflies were as follows :—

Species of *Phlebotomus*	Total No. examined	Reaction positive	Reaction negative	Per cent. positive to human serum
papatasii	367	196	171	52·5
chinensis	48	23	24	56·5
sergenti	28	18	10	55·5
caucasicus	32	14	18	57·1
major	1	1	0	0
borovskii	1	0	1	0
Undetermined ...	19	8	11	(?)

It is seen that more than 50 per cent. of the species of sandflies commonly found in Tashkent feed on human blood, from which it can be assumed that they are equally anthropophilic and zoophilic in their feeding habits. *C. A. Hoare.*

LEPROSY.

Doull (James A.), Rodriguez (José N.), Guinto (Ricardo) & Plantilla (Fidel C.). **A Field Study of Leprosy in Cebu.**—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 141–170. With 4 text figs. & 11 figs. on 3 plates.

The Philippine Island of Cebu has only one-twelfth of the total population but has produced one-quarter of the registered lepers, so a portion of it was selected for a field study of the distribution of the disease. A skin dispensary had previously been started to gain the confidence of the people, and very few of them objected to a practically complete examination of their bodies for lesions with special reference to household and family infections. The climate is humid, the people poor and the diet mainly of fish. No less than 5,987 of the 6,063 registered population were seen, or 98·3 per cent., and 104 leprosy cases, or 1·72 per cent., were found, or if apparently quiescent or arrested cases are excluded, 1·34 per cent. ; including 30, or 29·0 per cent., unknown previously to the Bureau of Health, but only 3 of these were bacteriologically positive against 43 under previous control. A study of the location of the first lesions confirmed Rogers and Muir's statement that a large proportion, 91 per cent., occur on the extremities exposed to injury. The age distribution supported the view that early infection is the rule and that males suffer most in adult life, but not in childhood ; this is most readily explained by greater exposure of males to infection after childhood. Previous contact with a leprosy patient, at least as intimate as sleeping in the same house, was met with in 38·5 per cent., and family contact in 26 per cent. In all but two instances the antecedent leper is known to have been bacteriologically positive. House overcrowding predisposed to infection, especially as regards sleeping accommodation. There was no serious deficiency of vitamin B as beriberi was rare. Yaws is very prevalent, but no difference was found in the leprosy incidence of yaws infected and other families, and no relation to the prevalence of other skin diseases. If a similar relationship between the segregated and the total cases obtains in the rest of the Philippines the total would be about 20,000. *L. Rogers.*

Holland (E. M.). **Leprosy in New Guinea.**—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 171–176. With 1 fig.

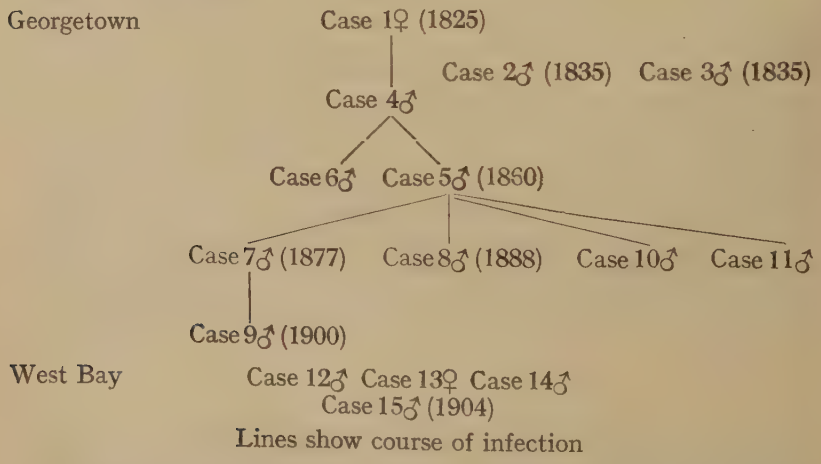
A few lepers were known in this area in 1914, a focus was found in the Madang District in 1926 ; the author found 32 cases in New Ireland in 1928, and by 1935 there were 547 such patients with an incidence of 11 per mille in New Ireland, 59 per mille in New Hanover, and 200 per mille in the earliest infected villages ; mostly mild, for 90 per cent. were nerve cases. In 10 per cent. the lesions appeared in childhood, and the majority about puberty or early adult life. These data were obtained by trained patrolling officers examining the whole population completely every few months. Treatment by alepol and moogrol has cleared up 52 of 546. *L. R.*

Mackenzie (J. Noble). **A Note on Leprosy Work in Korea.**—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 215–218.

The well-known institution at Fusan in Korea now has almost 600 patients and 22 children of lepers. The case mortality has been reduced by injections of hydnocarpus oil with 1 per cent. creosote from 25 to 2 per cent., and 60 to 70 recovered patients are being discharged annually. The Japanese Government have recently notified that all leper institutions must be registered under the rules for the isolation of infective diseases. At present not more than 6,000 of the estimated 20,000 in Korea are hospitalized. The author thinks that the greater proportion of known male to female lepers is due to the latter keeping going and the disease at bay longer. *L. R.*

Leprosy Review. 1936. July. Vol. 7. No. 3. pp. 101–150. With 9 figs. & 1 map on 6 plates & 2 diagrams.—Quarterly Publication of the British Empire Leprosy Relief Association, 131 Baker Street, London, W.1. [2*s.*]

A report on leprosy in the Cayman Islands by the Medical Officer (unnamed). The disease is stated to have made its appearance about 1825 and its subsequent history is recorded, with the following interesting diagram tracing the infection of fifteen cases from one patient to another down to 1904.



Lines show course of infection

An abstract of the report of Fiji for 1934 states that in the leper island of Makogai the cases from various British Pacific Islands receive treatment and care unsurpassed in the world. [This affords a good example to the isolated efforts of the smaller British West Indian Islands.] The incidence in different islands varies from one to two per cent. of the population. The admissions numbered 89, half of them nerve cases, only two with deformities, and none advanced cutaneous ones ; this is due to the importance of early diagnosis and treatment being now realized by doctors and patients. The standard treatment is weekly intramuscular injections of chaulmoogra oil with 0·5 per cent. of iodine, warmed and kept in a thermos flask or in glass vessels standing in hot water to reduce its viscosity, and this much cheaper preparation

gave as good results as ethyl esters. Recoveries numbered 29 in the year. The patients do much towards growing their food supplies, including fowls and their eggs. In spite of damage by storms about 100 *H. wightiana* trees supply about three pints of oil, but the *H. anthelmintica* are less healthy and have not yet borne fruit.

Brief reports from several British African Colonies are also summarized in this issue. *L. R.*

Binford (C. H.). **The History and Study of Leprosy in Hawaii.**—*Public Health Rep.* 1936. Apr. 10. Vol. 51. No. 15. pp. 415–423. With 4 figs. on 2 plates.

This note relates the oft-told tale of leprosy in the Sandwich Islands, and the prolonged attempts to reduce it by means of compulsory isolation, the influence of which is said to have been very doubtful. *L. R.*

Leprosy in India. 1936. Apr. Vol. 8. No. 2. pp. 43–91. With 6 figs. on 3 plates. Issued quarterly by the Indian Council of the British Empire Leprosy Relief Association.

The only original paper in this number is an address to the Mission to Lepers Conference in Calcutta on February 6th, 1936, by Dr. J. Lowe on Modern thought on Leprosy and its bearing on Mission work in India. He considers that although no one advance of major importance has been made recently, yet a number of bits of knowledge put together have greatly altered for the better the aspect of the leprosy problem. The optimism regarding treatment that began about twenty years ago has been replaced by a swing of the pendulum, perhaps too much, in the other direction. The isolation of infectious cases, particularly from young children is of prime importance. In Japan separation of children from their parents, and sterilization of patients, are regarded as very important measures. Quality rather than quantity of measures should be aimed at. Further investigations on treatment and epidemiology are required. The mission leprosy institutions are making a valuable contribution to anti-leprosy work in India. *L. R.*

Maxwell (James L.). **The Task before us.**—*Chinese Med. Jl.* 1936. May. Vol. 36. No. 5. pp. 716–720.

This is an address, on very similar lines to the above, on the leprosy position in China, where the problem is a most difficult, but not an insoluble, one. Surveys to throw light on the distribution and epidemiology of the disease are urgently needed. " It must be made clear to our legislators that penal laws are no more suitable for leprosy than for any other disease and that the use of force will tend to spread not to limit leprosy." He advised the formation of a Council on Leprosy by the Chinese Medical Association. *L. R.*

Mora (A. Damas) & Soares (J. C.). **Leprosy in Macao. Some Comments by the Portuguese Doctors in Macao.**—*Chinese Med. Jl.* 1936. May. Vol. 50. No. 5. pp. 721–725.

According to this account the Portuguese established an asylum for lepers in the Chinese port of Macao as early as 1569, and at the beginning of the 17th century the inmates numbered about 70. In 1930 a

separate new institution was built for women, but it could not be completed because no one would take on the work when the contractor developed leprosy. Among 150,000 people police search discovered 50 to 60 patients, who are receiving the usual treatment. *L. R.*

PEREIRA (Loiola). **Contribution to the Study of Leprosy in Goa.**—Reprinted from *Med. Digest.* 1936. Mar. Vol. 3. No. 12. pp. 540–542. With 1 chart.

This brief note records that the age incidence of leprosy in Goa was found to be higher than usually reported; for only 16·68 per cent. showed the first signs by the age of 20 years, nearly one-fourth at 31 to 35, more than half between 21 and 40 years of age, and two-thirds between 21 and 45. *L. R.*

INNES (James Rose). Occupation and Leprosy.—*Med. Jl. Australia.* 1936. June 13. 23rd Year. Vol. 1. No. 24. pp. 815–818. [14 refs.]

HURWITZ (Ezra) & ANDERSON (Hamilton H.). **Leprosy in Panama. First Thirty Years of Segregation.**—*Amer. Jl. Trop. Med.* 1936. May. Vol. 16. No. 3. pp. 353–369. With 1 chart. [13 refs.]

Since 1904, 320 leprosy patients have been segregated in Panama, half of whom were born in other areas of the Caribbean, and only three were born in the Canal zone. Only 33 children have been admitted. Fifteen arrested cases prefer to remain in the colony. None of the 17 uninfected persons employed in the colony up to as long as twenty years show any signs of leprosy. Forty-four patients have been paroled by a board of three members, 142 have died, 18 absconded, 18 have been repatriated and 12 readmitted since 1904. Tuberculosis and nephritis were the commonest causes of death, as usual. The average age of onset was 31 years and the duration of the disease on entering the colony 3·7 years. The duration of the disease in the present group of 108 has averaged 9·84 years to date. Esters are now being used, mainly intradermally. Cases improving under chaulmoogra therapy show lower unsaturation and higher total lipoids in their blood than those who are quiescent or deteriorating. *L. R.*

MEDEDEELINGEN VAN DEN DIENST DER VOLKSGEZONDHEID IN NEDERLANDSCH-INDIË. 1936. Vol. 25. No. 1. pp. 1–95.—[**15 papers on Leprosy in the Netherlands Indies.**]

SITANALA (J. B.) & KODYAT (R.). Lepra-Untersuchungen auf Java. [**Leprosy Investigations in Java.**]—*Arch. f. Dermat. u. Syph.* 1936. May 15. Vol. 174. No. 2. pp. 143–165. With 8 figs.

——. Leprabekämpfung. [**Leprosy Control.**]—*Ibid.* pp. 182–189.

This series of papers on leprosy in the Dutch East Indies is largely of local interest. The most important is one on the present Government policy. In 1897 they accepted the finding of the first International Leprosy Conference in favour of the principle of compulsory segregation, but it has never been legally enforced, and a Bill of 1925 making it possible was rejected. In 1930 the Leprosy Commission of the League of Nations at Bangkok under the heading of "Principles of the prophylaxis of leprosy" concluded: "In the present state of knowledge regarding leprosy prophylaxis the most important line of attack, in

addition to that by isolation, is by treatment—where efficient treatment is possible, segregation may be made less rigorous on the condition that it is supplemented by out-patient treatment." The Manila Leprosy Conference of 1931 endorsed this principle and "the leprologists assembled at Manila regarded the compulsory segregation of lepers to be a merely secondary consideration." The Netherland Indies Government officially recognized this new course of action in 1930–31, and a program drawn up by SITANALA is now being put into practice under him. The Queen Wilhelmina Jubilee Foundation is employing its funds on scientific leprosy research in a special institute, and a leper section and laboratory have been established in a Central Civil Hospital. Early cases are sought out and treated, especially in the young. Infective cases are being isolated locally at the expense of their communities. In short, modern methods based on recently acquired knowledge are being adopted.

Surveys have shown 3 per mille cases in Celebes, 3 to 9 per mille on Small Kei Island, and up to 17 per mille in mountain villages. *L. R.*

GERMOND (R. C.). **A Study of the Last Six Years of the Leprosy Campaign in Basutoland.**—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 219–224. With 3 figs.

This paper adds little to the recent one of P. D. STRACHAN (this *Bulletin*, 1936, Vol. 33, p. 295). The addition of further trained inspectors is believed to mark the beginning of a new and more hopeful era, for the admissions increased steadily between 1929 and 1933, with a slight decrease in 1934. More important, there has been a great shortening of the duration of the disease before admission, for in 1924 in only 31 per cent. was it one year or less, and in 37 per cent. over two years, against 57 and 25 per cent. respectively in 1934. The great majority of new cases now belong to the milder neural and tuberculoid forms. *L. R.*

WAYSON (N. E.). **The Early Diagnosis of Leprosy. The First Clinical Findings observed in Segregated Children of Leprous Parents.**—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 177–188. With 8 figs. on 2 plates.

This is an interesting account of the closely observed earliest symptoms in 108 children, nearly all of whom had been removed from their leprous parents at birth or soon after. Their ages varied from a few months to sixteen years, and thirty-five of them were found to exhibit at some time one or more of the changes that are considered to be possibly early signs of leprosy, but which may disappear again in some cases. X-rays may be helpful in detecting early bone changes. Of the 35 the suggestive changes in 25 have so far failed to develop any sensory disturbances, or any combination of changes which would justify a definite diagnosis of leprosy; but 10 have developed such changes. One, a boy of ten, is positive bacteriologically, with typical skin and nerve lesions.

"Among the remaining nine cases the following clinical findings have been obtained, either at the first examination or subsequently, in the numbers of patients indicated: Lagophthalmos, 8; droop of an angle of the upper lip, 6; atony or slight atrophy of the muscles of the interosseous spaces or of the thenar or hypothenar eminences, 6;

paresis or paralysis of the lumbrical or interosseous muscles of the hand, 1 ; thickened nerve trunks or branches, auricular, ulnar, or peroneal or (in one case) supraclavicular, 9 ; trophic disturbances, dryness or anhydrosis, scaliness, glossiness or wrinkling, in one or more areas of the skin other than in definitive lesions, 5 ; sensory disturbances in one or more areas of the skin without definitive lesions, 5 ; circumscribed or definitive skin lesions (macules), 4 ; sensory changes in one or more macules, 4."

The diagnosis was only made in these from a combination of findings including sensory disturbances. In four of the nine typical bacteria were demonstrated after several attempts. *L. R.*

Lefrou (G.) & Des Essarts (J. Quérangal). Contribution au diagnostic des faux lépreux. Les macules de vitiligo. [**The Diagnosis of Lepra-like Conditions. Leucoderma.**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 459–464.

This note deals with the differentiation of leprosy lesions from leucoderma. Stress is laid on the absence of anaesthesia, and lepra bacilli in the latter, or any thickening of the skin that can be felt with the eyes shut, as well as the absence of any inflammatory changes microscopically. *L. R.*

Tajiri (Isamu). **Leprosy and Childbirth.**—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 189–194. With 4 figs. on 1 plate.

It is commonly held that leprosy is liable to become aggravated during pregnancy and in the puerperal period, but few actual data are available on the subject. The author furnishes these in relation to the birth of 112 children to leprosy infected mothers in the Okayama National Leprosarium of Japan. His conclusions are well summarized as follows :—

" 1. Of 112 leprous women at Aisei-en who have had children, 39 (34·8 per cent.) developed the disease during pregnancy or shortly thereafter.

" 2. In 100 pregnancies that occurred in women with leprosy, exacerbation of the disease occurred 48 times, the remaining 52 being uneventful in this respect.

" 3. Though the initial symptom of leprosy is usually a simple macule, or anesthesia, the symptoms that appear in pregnancy or childbirth are usually acute lesions, the so-called " rash," often appearing as numerous active macules and frequently with edematous, erysipelas-like swellings of the face.

" 4. In the case of abortion there usually is little advance of the disease, though occasionally a case becomes worse in spite of it.

" 5. It is evident that for women who are in the incubation stage of leprosy, pregnancy and childbirth are liable to precipitate the development of the disease, and for those who have leprosy childbearing is apt to lead to its exacerbation and extension." *L. R.*

Berny (H.). Quel est l'avenir des enfants nés de mères lépreuses ? [**The Future of Infants born to Leprous Mothers.**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 469–471.

Girard, in 1933, showed that only very early separation from their leprous mothers will save infants from infection. Hasseltine in Molokai met with no infections among 109 infants rapidly removed after birth from the leper colony, but of those who lived in contact with their

mothers no less than 56 per cent. became infected. HOLMANN reported 4 per cent. of infections after 4 years 8 months, and 15 per cent. after 7 years 6 months residence with their infected mothers. The author records that three infants removed at birth from mothers suffering from fully developed nodular leprosy remained healthy, but 40 per cent. of ten infants remaining with such mothers developed the disease, and two of four infants of earlier dermal leprosy patients were also infected. On the other hand, three infants of nerve type mothers remained healthy. *L. R.*

ALEIXO (A.). Lepra e traumatismo. [**Leprosy and Trauma.**]—*Folha Med.* 1936. July 15. Vol. 17. No. 20. pp. 277–285. With 3 figs. [59 refs.]

The author discusses the subject from several aspects and includes a few illustrative cases showing how the disease in a state of latency may be unmasked (desvendada) by injury at a site where there are no apparent leprous lesions and where the bacilli cannot be found; secondly, how injury of a leprous lesion, in which bacilli are known to be present, may extend the infection locally and by the lymphatics bring about wider diffusion ; thirdly, accidents or complications, ocular conditions, deformities of the extremities, may be attributed to the trauma when they are in reality due to the leprosy itself [this may be so primarily, but it would not be easy to differentiate the part played by trauma on a site perhaps anaesthetic owing to the leprosy]. Lastly, the situation and nature of a lesion may lead the physician to a diagnosis of leprosy where no bacilli can be demonstrated to confirm this, and further investigations show the non-leprous nature of the condition. Two or three examples of this are given, one of which may be cited where a man sustained an injury to his right hand which resulted in anaesthesia of the ulnar aspect of the hand and of the fingers. He presented himself with an ulcer of the thumb which started in an injury 8 days previously. Leprosy was strongly suspected, but no bacilli could be found and both lesions were explicable by trauma alone.

H. H. S.

JUNIOR (Rabello). Sarcoide de Boeck leprogenico. [**Boeck's Sarcoid in Leprosy.**]—*Rev. Brasileira Leprologia.* S. Paulo. 1936. June. Vol. 4. No. 2. pp. 123–136. With 19 figs. [35 refs.]

Is Besnier-Boeck's disease a condition distinct aetiologically from leprosy? The author is of opinion that at present it would be premature to give a categorically positive reply. It *might* possibly be due to a virus, as yet unknown, producing epithelioid granulomata resembling tuberculomata in tending to caseation and lepromata by their neurotropism. Meanwhile the following epitomizes our present knowledge of the subject :—

1. In leprosy sarcoid lesions of the skin and other tissues occur.
2. Leprosy seems with peculiar frequency to determine the site of these granulomata.
3. In leprosy a rhinitis may be set up indistinguishable from sarcoid of the same site and bacilli may be looked for in vain [this would seem rather begging the question ; if the two here are not distinguishable, how can the author maintain that there are two conditions "resembling each other"?].

4. Leprosy is in a very large proportion of cases [the author states 95 per cent.] associated with indolent adenopathy analogous with sarcoids and histologically similar.

5. Pulmonary and bony lesions occur in leprosy radiologically indistinguishable from sarcoids.

6. Leprosy, even in its florid forms, is accompanied by cutaneous allergy to tuberculin, and the same holds good in the " so-called Besnier-Boeck disease " [there is no reason why both should not react to tuberculin].

7. Leprosy, especially the tuberculoid forms, benefits from antileprol as does sarcoid. *H. H. S.*

Panja (Ganapati). Two Cases of Leprotic Abscess.—*Calcutta Med. Jl.* 1936. Apr. Vol. 30. No. 10. pp. 605–606. With 1 fig.

Muir (E.) & Chatterji (S. N.). **A Study of Nerve Leprosy.**—*Indian Jl. Med. Res.* 1936. July. Vol. 24. No. 1. pp. 119–138. With 6 plates.

This illustrated study of the history and clinical conditions, with pathological and bacteriological examinations of portions of removed tissues, in eighty-one cases of leprosy cannot better be summarized than in the authors' own conclusions. It should be read in the original by all interested.

" 1. Bacilli enter and pass up nerve branches from the neuro-vascular plexuses of the skin. This may, or may not, be accompanied by clinical skin lesions. Either the local branches, or the distant main nerves, or both, may show clinical changes. Bacilli lie in close connection with neuro-vascular plexuses in the skin and subcutaneous tissue. The nerve bundles form a specially favourable medium for the growth of *M. leprae*.

" 2. Bacilli are carried up the nerves from the skin by the lymph flow. They tend to accumulate at points of obstruction.

" 3. The ' neurotropism ' of *M. leprae* is discussed. This is shown to be at least partly connected with the comparative absence of cellular response to the bacilli in the nerves.

" 4. The endothelial cells of the capillaries take the most important part in the response to *M. leprae* in both the skin and the nerves. In the latter the bacilli are isolated from the endothelial cells of the centrally placed capillary by the medullated nerve fibres, and thus may escape phagocytic destruction. In the skin the bacilli lie in close proximity to the endothelial cells, and are thus more liable to be phagocytosed.

" 5. The effects of varying degrees of cellular reaction to lepra bacilli in the skin and in the nerves are discussed, especially with regard to the formation of nerve abscess.

" 6. Apart from the degree of local concentration of bacilli, there are three factors which account for varying degrees of resistance and cellular response to *M. leprae*, viz., specific resistance, general health and allergic reaction.

" 7. The course of events is traced in the formation of caseation and nerve abscess ; the forms which nerve abscesses assume are described and discussed in relation to their effect on nerve functions.

" 8. The nerves form an important reservoir for bacilli where they may multiply until sufficient tolerance is produced to overcome the greater resistance in the skin. This is followed by spreading of the bacilli to other nerves, and frequently by generalized infection.

" 9. Pain and other symptoms are relieved in suitable cases by excision of the nerve sheath of thickened nerves, opening of abscesses and excision

of diseased nerve branches. A distinction is made between primary and secondary anaesthesia.

" 10. ' Recovery reaction ' is distinguished from allergic reaction."

L. R.

GRIECO (Vicente). Estudo clinico e histologico de um caso de nevrite hanseniana tuberculoide com caseificação e ulceração (abcesso de nervo). [**Clinical and Histopathological Study of a Case of " Nerve Abscess " in a Leper.**]—*Rev. Brasileira Leprologia.* S. Paulo. 1936. June. Vol. 4. No. 2. pp. 151–201. With 35 figs. [18 refs.]

This is a very minute and detailed study of the case of a leper, 24 years of age, with a lesion in the lower third, antero-external surface, of the right leg. The lesion consisted of two ulcers about 4 cm. apart, and on the dorsum of the foot was an anaesthetic macule. The ulcers were in relation to the musculo-cutaneous nerve ; this nerve-branch was thickened and passed on to the site of the macule. In addition to chaulmoogra oil treatment, surgical intervention was undertaken and the nerve extirpated from the upper level of the swelling down to the macule. Now, a year later, there has been no further sign of the disease.

A large number of serial sections was made, stained and examined, and all stages could be made out, from an early proliferation of histiocytes and lymphocytic infiltration among the nerve fibres to the advanced stage of caseation. *H. H. S.*

HOFFMANN (W. H.). Lepra und Bananen. [**Leprosy and Bananas.**]—*Muench. Med. Woch.* 1936. May 8. Vol. 83. No. 19. pp. 775–776.

Anxiety has been expressed concerning the possibility of leprosy infection being acquired in Germany through imported bananas.

The author considers the question and comes to the conclusion there is absolutely no basis for such an idea. *L. R.*

KRIZ (J. R.). **Biological Study of " R " and " S " Forms of Chromogenic Acid-Fast Bacillus from Human Leprous Lesion.**—*Proc. Soc. Experim. Biol. & Med.* 1936. Apr. Vol. 34. No. 3. pp. 303–306.

The author has studied the variations in the colonies of an acid-fast bacillus isolated from a subcutaneous leprosy nodule and cultivated by the method of Duval. He separated " S " or smooth from " R " or rough forms. The " S " forms show smaller moist colonies, are acid-fast rods with 2–5 small granules, which he considered to be signs of degeneration. The " R " forms show larger wrinkled dry colonies, grow more rapidly, form a flaky suspension and are more thermostable. They also show mainly large bipolar granules resembling a spore stage, and the bacilli are surrounded by a thicker capsular zone. Both forms may remain pure on subculturing over several months, but the " S " form may revert to the " R " during adaptation on artificial media, and it must be assumed that they are reversible. The " S " is considered to be the typical and the " R " the mutant form. *L. R.*

Villela (Gilberto G.), Castro (Almir) & Anderson (Jeanette V. D.). Lipemia na lepra. [**Lipaemia in Leprosy.**]—*Rev. Brasileira Leprologia.* S. Paulo. 1936. June. Vol. 4. No. 2. pp. 111–120. With 3 graphs. [21 refs.]

The authors examined a hundred lepers showing the cutaneous, the nervous and mixed forms, and ranging in age from infancy to 40 years, and both white and coloured patients. They found that total lipids and fatty acids were above the normal in all, whereas the cholesterol was in general lowered. Neither the type of disease nor the treatment influences the lipaemia to any appreciable extent. As a general rule the whites showed higher levels than the coloured patients. *H. H. S.*

Villela (Gilberto G.), Castro (Almir) & Anderson (Jeanette van D.). Blood Lipid Studies in Leprosy.—*Jl. Trop. Med. & Hyg.* 1936. June 1. Vol. 39. No. 11. pp. 126–127.

Villela (Gilberto G.) & Castro (Almir). Le cholestérol et ses fractions dans le plasma des lépreux.—*C. R. Soc. Biol.* 1936. Vol. 123. No. 28. pp. 433–435.

Anderson (R. J.), Crowder (J. A.), Newman (M. S.) & Stodola (F. H.). **The Chemistry of the Lipids of Tubercle Bacilli. XLIII. The Composition of Leprosin.**—*Jl. Biol. Chem.* 1936. Apr. Vol. 113. No. 3. pp. 637–647. **XLV. Isolation of α and β Leprosol.**—*Ibid.* June. Vol. 114. No. 2. pp. 431–439.

These two highly technical papers are best summarized in the authors' own words :—

XLIII. " Leprosin, a neutral wax-like substance isolated from the leprosy bacillus, has been analyzed and found to consist of a complex mixture of solid glycerides and waxes.

" The fatty acids liberated on saponification were myristic, palmitic, stearic, tetracosanic acids and a new hydroxy acid called leprosinic acid.

" The neutral portion of leprosin after saponification consisted of both water-soluble and ether-soluble components. Glycerol was the only water-soluble substance that could be detected.

" The ether-soluble unsaponifiable matter consisted of two higher, secondary, optically active alcohols. The less soluble alcohol was identified as *d*-eicosanol-2. A second alcohol, probably *d*-octadecanol-2, was also isolated."

XLV. " Two new alcohols, α- and β-leprosol, having phenolic properties, have been isolated from the unsaponifiable matter of the neutral fat from *Bacillus leprae* and some of their properties have been determined."

L. R.

Chorine (V.) & Crougue (O.). La cholestérine sanguine dans la lèpre murine. [**Cholesterine in Leprous Rats.**]—*C. R. Soc. Biol.* 1936. Vol. 122. No. 21. pp. 621–624.

Most workers have reported finding a decrease in the cholesterine in the blood of leprosy patients, but Marchand found it normal in early cases and Borgatti found it to be increased. The authors have therefore investigated the changes in the case of rats infected with murine leprosy, both naturally and by inoculation, and found only slight

variations from the normal limits, although the figures were somewhat lower in infected than in healthy animals. Thus the average in normal rats was 0·65 against 0·57 in infected ones, but the difference is of very little practical value, as the diminution is far too inconstant to be of any diagnostic importance and shows no definite relationship to the severity of the infection present. The figures in some diseased rats may be as high as in healthy ones. *L. R.*

Maldonado (L. García). La reacción de Kahn en la lepra. [**The Kahn Reaction in Leprosy.**]—*Gac. Méd. de Caracas.* 1935. Dec. 31. Vol. 42. No. 24. pp. 380–381 ; 1936. Jan. 15 & 31. Vol. 43. Nos. 1 & 2. pp. 9–16 ; 27–28.

Three significant cases are recorded : (1) A man of 20 years, with iritis of the right eye. There was no history of syphilis but a Kahn test gave 4 plus. Intensive antisyphilitic treatment had no effect on the reaction, and further examination revealed distinct evidence of a mixed form of leprosy—thickened ulnar nerves, zones of anaesthesia, falling off of the eyebrows, bacilli in nasal mucus. (2) A woman of 20 years, with papular lesions of the face. Kahn 4 plus, no improvement following two courses of antisyphilitic treatment. Leprosy bacilli found in large numbers in the papules. Intensive treatment with chaulmoogra brought about improvement in the clinical condition but did not affect the Kahn reaction. (3) A woman of 25 years, with epistaxis and " various nasal troubles." She was treated for syphilis, and the Kahn, 4 plus, was held to confirm the diagnosis. Two courses of arsenicals neither benefited the local condition nor changed the Kahn reaction. The author suspecting lepra examined some infiltrations which had appeared and found the bacillus. Chaulmoogra resulted in marked improvement, but the Kahn was still 3 plus.

He proceeded to examine many lepers in Providence Island as regards their Kahn reaction, and came to the conclusion that there at any rate the reaction was frequently positive even where syphilis and yaws could be excluded, and he sums up the question in rather obvious terms [reminiscent of the judge in Bardell *v.* Pickwick] : If in a leper the positive Kahn becomes negative under antisyphilitic treatment and at the same time the lesions improve, the reaction is to be regarded as due to the syphilitic element ; if the lesions remain unchanged, but the reaction becomes negative under anti-syphilitic treatment, both diseases are present ; if the Kahn becomes negative under treatment for leprosy, we ought to regard the reaction as having been due to the leprosy and not in any way the result of syphilis or yaws. Intermediate reactions are analogously interpreted quantitatively, whether reduced or not as the result of treatment. *H. H. S.*

Lai (Daniel G.) & Chen (Wen Ying). **The Wassermann and the Kahn Reactions in Leprosy.**—*Chinese Med. Jl.* 1936. May. Vol. 50. No. 5. pp. 702–704.

The authors record 12 per cent. positive Kolmer-Wassermann and 21·5 per cent. Kahn positives in 167 ambulatory leprosy cases, but 40 per cent. positive Kahn tests in 78 Hangchow leprosarium patients. The complexity of the factors prevents them reaching any definite conclusion, so further work is required. *L. R.*

LEFROU (G.). La valeur de la réaction de Vernes péréthynol dans la lèpre. [**Vernes Perethynol Reaction in Leprosy.**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 464–468.

The author records 188 observations on this test in lepers in Guadeloupe. He came to the conclusion that the flocculation of the serum by the perethynol is completely independent of the leprous factor, in spite of the fact that protein want of balance may exist in both leprosy and syphilis. This does not make the test useless, for it is important in treatment to know if leprosy is complicated by syphilis. *L. R.*

PEREIRA (Paulo C. R.). La réaction de fixation du complément avec l'antigène de Witebsky, Klingenstein et Kuhn dans la lèpre. [**Witebsky, Klingenstein and Kahn Tests in Leprosy.**]—*Internat. Jl. Leprosy.* Manila. 1936. Apr.–June. Vol. 4. No. 2. pp. 207–214.

The author reports reaction in the blood sera of 107 cases of leprosy. The reaction is a form of complement fixation ; first applied by its discoverers to tuberculosis, and later found by BRANTS in Riga and AOKI and MURAO in Japan to be present in a very large proportion of leprosy cases. The antigen is an extract of the tubercle bacillus, for the details of the preparation of which this paper may be consulted. The test proved positive in 100 per cent. of 12 of the cutaneous type, and in 35 mixed cases, as well as in 80 per cent. of 60 neural cases, although it was less strong in the latter. Among 84 sera of persons in continued contact with lepers, mostly children or mates of them, 53 gave negative, 4 doubtful and 27 positive reactions. Of the 27 positive all but 9 were probably leprotic or tuberculous, and the last require to be watched. This test, therefore, gives the highest number of positive reactions in all forms of leprosy of any, so it should be employed in cases of difficult diagnosis and in latent leprosy. *L. R.*

DUBOIS (A.). La réaction de Mituda. (Notice complémentaire.) [**Mituda's Reaction.**]—*Bull. Soc. Path. Exot.* 1936. June 10. Vol. 29. No. 6. pp. 649–651.

The author has obtained very similar results from this test to those of others. Heated and phenolized emulsions of lepromas are injected in 0·5 cc. doses and a papular reaction sought for after 8 to 20 days. The reaction is strongest in normal subjects and in leprosy cases with few bacilli, but negative in those showing many bacilli. The author injected 29 patients with 0·1 cc. intradermally and examined after 10 and 15 to 20 days : he obtained 5 strong, 10 moderate and 7 feeble reactions, but is able to come to no clear decision. *L. R.*

VAN BREUSEGHEM (R.). Etude de la réaction déterminée par la léprine de Loewenstein chez le lépreux et chez l'homme sain. [**Loewenstein's Leprine Reaction.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 109–113.

The author has used Loewenstein's leprine in lepers and the healthy by subcutaneous injection ; he obtained reactions in 39 per cent. of lepers and in about 62 per cent. of non-lepers. *L. R.*

Fraser (N. D.). **The Iam Tsau Clinic for Patients suffering from Leprosy.**—*Chinese Med. Jl.* 1936. May. Vol. 50. No. 5. pp. 693–701.

This is an interesting account of a Chinese leprosy clinic founded by a Christian community and partly financed by the patients. It is an early response to recent Canton legislation authorizing the establishment of out-patient and village clinics for the treatment of leprosy patients. The Swatow district is estimated to have 10,000 lepers, and it is hoped that the success of this clinic will lead many more to be opened. At the opening in September 1934, 26 patients attended, at the end of six months 128 had been registered and 99 are attending fairly regularly in spite of some having to come from far, with 50 to 75 per cent. of possible attendances. Neural cases form 50 per cent., and some 29 per cent. are fairly early cases. Alepol and iodized moogrol are used, and some nodular cases responded rapidly to intramuscular and intradermal injections. The cost has been 25 cents. per visit. It is too early to speak of results, but a promising start appears to have been made. *L. R.*

Tolentino (José G.). **Certain Factors supposed to influence the Results of the Treatment of Leprosy.**—*Philippine Jl. Sci.* 1936. Feb. Vol. 59. No. 2. pp. 163–175.

This is a general discussion on the influence certain factors are supposed to have on treatment, based on the records of 477 cases treated at the Eversley Child Treatment Station in the Philippines between 1929 and 1933; apparently the more favourable earlier cases after 788 patients had been sent to the Culion Leper Colony or had died or absconded.

"1. The effect of certain factors supposed to influence the results of treatment has been studied.

"2. The severity of skin lesions was found inversely proportional to the percentage of paroled cases.

"3. The duration of the disease was found to have apparently no relation to the results of treatment.

"4. The age on admission was found to have a definite relation to the results of the treatment, in that the first, fourth, fifth, and sixth decades gave higher percentages of paroled cases than the other decades. The results, if plotted with age, gave a curve that goes down from the first to the third as the lowest point, gradually rising to the fifth decade as the highest point from which it goes down again.

"5. The results of the treatment were found to be apparently better among the females than among the males, except in the fifth decade, probably due to the menopause which disturbs the physiological life of the females.

"6. Contrary to what is commonly claimed, puberty and childbirth did not seem to be important factors in the treatment among these cases.

"7. The results of treatment were found to be good among the widowed patients, but this is due to the fact that they are generally of mature age and, in the groups studied, there were more females and more of the less-advanced cases than among the unmarried and married cases.

"8. The degree of nutrition was found to have a definite relation to the results of treatment. The results are inversely proportional to the degree of obesity of the patients. It appears that excess fat in the body has an unfavorable effect on the treatment of leprosy." *L. R.*

VAN BREUSEGHEM (R.). Essai du 4828 A dans le traitement de la lèpre. [**Trial of 4828 A in Leprosy.**]—*Ann. Soc. Belge de Méd. Trop.* 1936. Mar. 31. Vol. 16. No. 1. pp. 115–119.

The author reports on eight cases of leprosy treated by an ethyl chaulmoograte of undisclosed composition called 4828 A. It was well tolerated, but without any special activity in the cases dealt with.

L. R.

DELANOE (E.). A propos du traitement mixte de la lèpre. [**Mixed Treatment of Leprosy.**]—*Bull. Soc. Path. Exot.* 1936. June 10. Vol. 29. No. 6. pp. 641–646.

The author records his experience in treating 150 cases of leprosy for comparatively short periods under favourable hospital conditions. As a result he advises mixed treatment with various combinations of chaulmoogra derivatives, arsenical preparations such as Novarsenobenzol and acetylarsan, B.C.G. Vaccine, solganal B, methylene blue, crisalbine, etc. *L. R.*

RYRIE (Gordon A.). **The Therapeutic Effects of Phthalic Acid Salts.**—*Internat. Jl. Leprosy.* 1936. Apr.–June. Vol. 4. No. 2. pp. 201–206.

A new water soluble salt of phthalic acid, called cotarnine, has been tried in 20 cc. doses of a 1 per cent. solution twice a week in 19 cases; with marked improvement during the first three months only in 9 cases, accompanied by a drop in the sedimentation rate. Three other phthalic acid salts produced no benefit. *L. R.*

MONTEL (R.). Conférence sur le traitement de la lèpre par le bleu de méthylène. [**Conference on Methylene Blue Treatment.**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 361–371.

——, BABLET (J.), NGUYEN NGOC NHUAN & DO VAN HOANH. Deux cas de lèpre traités par le bleu de méthylène seul, d'abord, et par l'association: "Bleu de méthylène-chaulmoogra," en suite. Action du traitement sur les symptomes cliniques, sur les tissus et sur le *Mycobacterium leprae.* [**Two Cases treated with Methylene Blue.**]—*Ibid.* May 13. Vol. 29. No. 5. pp. 560–576. With 10 figs.

The first of these papers records the following results of the treatment of 351 leprosy cases by combined chaulmoogra derivatives and methylene blue.

	Per cent.
Clinically cleared up	10·26
Notably ameliorated	34·76
Ameliorated	45·87
Stationary	6·55
Aggravated	1·42
Died	1·14

The second paper records in detail, with good photos, remarkable improvement in two fairly advanced nodular cases of leprosy, together with microphotos illustrating the tissue reactions. *L. R.*

Féron. Une indication du bleu de méthylène dans le traitement de la lèpre.—*Bull. Soc. Path. Exot.* 1936. June 10. Vol. 29. No. 6. pp. 646–647.

Gramberg (K. P. C. A.). Eenige beschouwingen over de praktijk der leprabehandeling en -bestrijding in N.O.I.—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. July 7. Vol. 76. No. 27. pp. 1700–1721. English summary.

Watanabe (Yoshimasa). **Experimental Studies on Animals concerning Leprosy. Report VI. Inoculation Tests with Rat Leprosy (Part 2).**—*Kitasato Arch. Experim. Med.* 1936. Apr. Vol. 13. No. 2. pp. 158–168.

The author records attempts to infect guineapigs, rabbits and monkeys by inoculating them subcutaneously and intratesticularly with emulsions of rat leprosy nodules. In the case of guineapigs the results were negative, although the acid-fast organisms may remain alive in their bodies for some time. In rabbits in some instances very mild slightly developed leprous changes were found at the site of inoculation with retention of the vitality of the bacilli for a time, but it can be said in a general way that normal rabbits are not susceptible to leprosy. In Japanese monkeys none of the animals showed clear evidence of complete infection, although large doses of rat leprosy material may produce temporary leprous changes containing numerous acid-fast organisms, but the lesions heal readily and spontaneously without leaving any signs of leprosy. *L. R.*

Lampe (P. H. J.) & de Moor (C. E.). Ratten-Lepra. [**Rat Leprosy.**] —*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. June 30. Vol. 76. No. 26. pp. 1619–1641. With 3 graphs. English summary.

The authors have made an extensive study of the incidence of rat leprosy in Batavia. Clinically they differentiate between lymphoid and skin leprosy, and found 930 of the former and 225 of the latter per 10,000 rats. The natural infection is a lesion of the subcutaneous lymph glands of the groin, jaw and mesenteric regions, and the bacilli are also regularly found in the skin at the root of the tail. The house rats are most affected. *R. concolor* and *R. norvegicus* suffer more than *R. r. diadii*. Infection probably occurs chiefly through skin injuries and cannibalism may account for the one-third of the cases in which the jaw and mesenteric lymph nodes are involved. *L. R.*

Prudhomme (R. O.). Le glutathion total dans la lèpre murine. [**Glutathion in Rat Leprosy.**]—*C. R. Soc. Biol.* 1936. Vol. 122. No. 22. pp. 739–741.

The author had previously found a notable diminution of glutathion in severely infected leprous rats, but normal figures in those slightly attacked. He now reports on the relationship between oxidized and reduced glutathion in infected rats and concludes that although the reduced substance falls in badly involved organs, the pre-existent glutathion is not oxidized but disappears. *L. R.*

Lobel (L. W. M.). Büffellepra (Lepra bubalorum). [**Buffalo Leprosy.**]—Reprinted from *Handelingen v/h 7de Ned. Ind. Natuurwetenschappelijk.* p. 385.

In buffaloes of the Netherlands Indies (*Bos bubalus* var. *sondaicus*) a skin disease occurs which has not been noticed elsewhere. So far 63 cases have been discovered, distributed as follows: In Java—Semarang 9, Cheribon 7, Serang 5, Batavia 2; in Celebes—Manado 37, Watampone (Makale) 2, Makassar 1.

In Ressort Paloe (Manado) leprosy of buffaloes is comparatively common. Search is being made in this district for cases in an early stage and to discover, if possible, whether there are any latent cases. At the Seventh Scientific Congress, clinical bacterioscopic and histological pictures of lepra bubalorum were shown on the screen.

H. H. S.

HELMINTHIASIS.

LEIPER (R. T.). **Crustacea as Helminth Intermediaries.**—*Proc. Roy. Soc. Med.* 1936. July. Vol. 29. No. 9. pp. 1073–1074 (Sect. Trop. Dis. & Parasit. pp. 43–44).

A demonstration by microscope and lantern of crustacea as helminth intermediaries.

There is need for regional surveys in the tropics of species of crustacea, specially of the Entomostraca and of the genus *Cyclops*. That *Cucullanus* underwent development in Cyclops led LEUCKART to suggest to FEDSCHENKO that *Dracunculus* did so. Of this FEDSCHENKO thought he had got proof, but LEIPER, contrasting the metamorphosed larva of *Dracunculus* which he had himself got in Cyclops with that of *Cucullanus* showed the likeness of Fedschenko's larvae to those of *Cucullanus*. Preparations showed the development of *Gnathostoma spinigerum* in English cyclops. *Cyclops* also harboured parasites of fish and turtle. None of man's trematodes has been shown to develop in Cyclops, but the pseudophyllidae need a *Cyclops* species as their first intermediary. No cyclophyllid tapeworm of man makes use of Cyclops.

Development of trematodes of man in crustacea is undergone only by *Paragonimus westermanii* in *Malacostraca*. The list of species concerned is :—

" *Astacus* (*Cambaroides*) *japonicus*, *A.* (*C.*) *similis*, *A.* (*C.*) *dahuricus*, *A.* (*C.*) sp., *Potamon* (*Geothelphusa*) *dehaani*, *P.* (*G.*) *obtusipes*, *P.* (*Parathelphusa*) *sinensis*, *Pseudothelphusa iturbei*, *Eriocheir sinensis*, *E. japonicus*, *Sesarma dehaani* (experimental : not a food crab)."

In discussion Arthur BLOK asked whether any European fresh-water mollusca acted as hosts of *P. westermanii* ; if so the mitten crab, the second intermediary, might have a sinister effect on health in Europe. In replying Professor LEIPER pointed out that mollusca were much more specific as first intermediaries than crustacea as second intermediaries and that none of the first intermediaries had been reported in Europe. He thought that the prohibition of infected Asiatics into the United States had a political rather than a parasitological significance.

Clayton Lane.

PETERS (B. G.). **Some Recent Developments in Helminthology.**—*Proc. Roy. Soc. Med.* 1936. July. Vol. 29. No. 9. pp. 1074–1084 (Sect. Trop. Dis. & Parasit. pp. 44–54). [64 refs.]

A picture of parasitism, as exemplified by cestodes under the heads of life-history anomalies, resistance to digestion, metabolism, immunity and immunological reactions.

The author concludes :—

" They repeatedly follow the complicated routine of their life-cycle, yet depart from it occasionally in an attempted adjustment to abnormal circumstances. They display ingenious processes, physical and chemical, for resisting the digestive ferments of the host and so gain access from the outside world to the host's tissues or intestinal lumen. Once there, they adapt themselves to changed conditions (plentiful food but little oxygen) by assuming an anaerobic mode of life. Then the host intervenes, at least in some somatic infections, by elaborating antibodies which protect it from super-infection. So that, in the end, something like equilibrium between the two is achieved. These antibodies, cautiously used, give the clinician a number of valuable diagnostic techniques.

"The picture is, of course, far from complete even with respect to existing knowledge (it omits particularly the many tissue reactions of the host); and there are extensive areas which cannot yet be filled in. But it is more detailed than would have been possible ten years ago. With further researches into the physiology and immunology of helminthic infections, it is likely that their diagnosis, treatment, and prevention will become increasingly precise and effective." *C. L.*

i. Komiya (Yoshitaka), Kawana (Hiroshi) & Tao (C. S.). **On the Prevalence of Helminthiasis among the Japanese and Chinese in the District of Shanghai. I. Results of Examinations of Helminths Ova among the Chinese Children and Students.**—Reprinted from *Jl. Shanghai Sci. Inst.* 1936. Mar. Sect. IV. Vol. 2. pp. 85–94.

ii. ——, —— & ——. **II. Results of Examinations of Helminths Ova among the Japanese and Chinese Servants and Cooks in Restaurants and Cafés.**—Reprinted from *Ibid.* Apr. Sect. IV. Vol. 2. pp. 95–107. [32 refs.]

iii. ——, —— & ——. **III. Results of Examinations of Helminths Ova among the Japanese Primary School Children.**—Reprinted from *Ibid.* May. Sect. IV. Vol. 2. pp. 117–138. [21 refs.]

iv. ——, —— & ——. **IV. Results of Examinations of Helminths Ova among the Japanese Students of the Girls High School, Commercial School and Tung Wen College.**—Reprinted from *Ibid.* pp. 139–154. [16 refs.]

v. Tajimi (Tai). **V. Results of Examinations for Helminths Ova among Japanese Kindergarten Children and their Family Members.** Reprinted from *Ibid.* June. Sect. IV. Vol. 2. pp. 169–183. With 1 plate. [17 refs.]

Helminthiasis in Shanghai as disclosed after examination by a hydrochloric acid, antiformin, ether method. The scope of the different papers is seen in their subtitles.

i. The numbers tested were—in primary schools 55, middle schools 169, Medical College 312. The percentages of infection in the three groups were—ascaris 20, 55, 48, trichuris 11, 37, 37, hookworms, 0, 5, 6, trichostrongylus 0, 1, 1, clonorchis 2, 2, 5, fasciolopsis 0, 2, 1. The figures in papers by other workers in different parts of China are noted and infections with 2 or more sorts of worms are listed.

ii. The numbers tested were—Japanese 109, Chinese 181. The numbers of the two races in whom infection was seen were—ascaris 35, 64, trichuris 57, 94, hookworms 23, 44, threadworms 1, 1, trichostrongylus 1, 1, clonorchis 23, 13, fasciolopsis 0, 2, metagonimus, 2, 0. The distribution is put out in tables by age and sex. As many as 5 infections were present in the same person.

iii. The numbers tested were 2,523. The percentages of infection were—trichuris 37, ascaris 15, hookworms 5, clonorchis 3. There were seen a few infections with enterobius, trichostrongylus, metagonimus and hymenolepis. In about a quarter there was more than one infection. No marked difference in growth was seen in those with or without parasites.

iv. In the faeces of 303 girls the numbers in which eggs were found were—ascaris 71, trichuris 113, hookworms 27, trichostrongylus 3, clonorchis 23, *Hymenolepis nana* 1, total infected 166.

v. The numbers are few but the belief is that infection is likely to be familial with the mother mainly responsible for dissemination. *C. L.*

Robertson (R. C.). **The Transmission in China of Helminths by Vegetables.**—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 418–422. With 10 figs. on 5 plates.

The possibility of contamination of raw vegetables by worm eggs about Shanghai is great.

Vegetables are fertilized by human or animal faeces and they are often washed " in a neighbouring creek or pond where the night soil commodes have been rinsed out in the morning or where the water has been contaminated by sewage in a number of other ways." The deadly effects of heating ascaris eggs are illustrated by microphotographs, but Robertson thinks that the suggestion of heating the fertilizer at the central depot is Utopian. [By natural decomposition, the Beccari process sterilizes by heat even in small quantities and produces a most valuable humus.] *C. L.*

Tao (C. S.). **Transmission of Helminths Ova by Flies.**—Reprinted from *Jl. Shanghai Sci. Inst.* 1936. Apr. Sect. IV. Vol. 2. pp. 109–116.

Carriage of worm eggs by flies may take place on their legs or in their intestines.

In 64 fly traps, which were emptied in all 189 times, 146,563 flies were caught, 71 per cent. being *Musca*, 13 per cent. *Lucilia* and 16 per cent. *Compsomyia*. Worm eggs were found in the liquid of the traps in 8 of the 64 places in which they were set—38 eggs in all, and, in detail, 24 ascaris, 6 trichuris and 8 hookworm. Of 206 flies caught near latrines and dissected, 5·8 per cent. had these 3 sorts of eggs in their intestines and none on their legs, but flies fed on parasitic ova do have them on their legs, generally about the " pulvillus." Yet it is thought that the weight of these eggs will make a fly less airworthy so that it cleans these off quickly and so carries them for only a short distance in this way, but takes eggs further afield in its intestine. But, on the other hand, flies pass faeces very soon after taking food [and so probably pass worm-eggs also]. *C. L.*

Chen (H. T.). **Parasites in Slaughter Houses in Canton. Part I. Helminths of Kwangtung Hogs.**—*Lingnan Sci. Jl.* (continuation of *Lingnaam Agric. Rev.*) 1936. Vol. 15. No. 1. pp. 31–44. With 17 figs. [16 refs.]

Helminths, seen in 100 slaughtered hogs in Canton, which have the greatest importance for medicine are in order of frequency :—*Ascaris lumbricoides* 40, *Fasciolopsis buski* 26, *Clonorchis sinensis* 1 (in stomach), *Fasciola hepatica* 1. A few *Ancylostoma caninum* were found once in the stomach, and a single sparganum in the intestine taken in with food. *C. L.*

Ryo (Sai). **A Survey of the Distribution of Helminthic and Protozoan Parasites among 10293 Inhabitants of Fushun.**—*Jl. Oriental Med.* 1936. June. Vol. 24. No. 6. [In Japanese pp. 1145–1152. English summary p. 81.]

Parasitic infestation is heavy among the population of Fushun. The subjects examined included all classes—general civil population,

prisoners, mine-labourers, farmers, schoolboys and people attending the outpatient department of the Mukden Medical College. The commonest parasite was Ascaris, found in 84 per cent., Trichuris next 47·9, Endolimax 43·6, hookworm 42·7, the relative proportions of *A. duodenale* and *N. americanus* being nearly 6 to 1. *E. coli* was seen in nearly half (49·4 per cent.), *E. histolytica* in 22·9 ; the latter was found in 30 per cent. of the Manchurian labourers, 21 per cent. of the general population and 14 per cent. of the schoolboys. Taenia occurred in 5 per cent. of Korean schoolboys. Other findings, not numerically stated, were *Trichostrongylus orientalis, Strongyloides stercoralis, Hymenolepis nana* and *H. diminuta, Metagonimus yokogawai, Clonorchis sinensis, Fasciolopsis buski, Dicrocoelium dendriticum,* Chilomastix and Trichomonas. *H. H. S.*

Hall (Maurice C.). **Problems of Parasitism in Hawaii.**—*Rev. Parasit., Clin. y Lab.* Habana. 1936. May–June. Vol. 2. No. 3. pp. 367–383.

After considering the local conditions which do or do not favour parasitism, Hall deals with the parasites, mostly helminthic, of man, poultry, dogs, sheep, goats, cattle and horses as they came to his notice in the course of a month's visit to Hawaii.

As to man's parasites special mention is made of *Fasciola hepatica* with 8 cases known there, of *Trichostrongylus colubriformis* identified by its eggs, of hookworms of both the common species (conditions being apparently favourable for an attack on this problem), of trichuris which are common and seemingly linked with appendicitis, of threadworms and ascarids whose real incidence is unknown, of *Taenia saginata* and *Hymenolepis nana,* the latter being apparently the commoner, and of amoebae. *C. L.*

Iglesias (Aurelio Sousa). El parasitismo intestinal en la población escolar de San Ramón y algunas sugerencias de órden profiláctico. [**Intestinal Parasitism among School Children of San Ramón, Province of Tarma, Peru.**]—*Rev. Méd. Peruana.* 1936. May. Vol. 8. No. 89. pp. 306–316. With 15 figs.

Only those children who had lived for six months at least in the district were examined, but the number was small, 82. Intestinal parasites of one kind or another were found in all. In many the infestation was heavy and three-quarters of them showed multiple parasitism. The commonest was *Trichuris trichiura* in 96·1 per cent., next *Ascaris lumbricoides* 59·4 per cent., then in order *Strongyloides stercoralis* 33·3, *Ancylostoma duodenale* 14·5, *Enterobius vermicularis* 3·1, *Taenia saginata* 2·2, *E. histolytica* 8·3, Trichomonas and Balantidium each 3·1 per cent. One child harboured Balantidium, *E. histolytica,* Ascaris, Ancylostoma and Trichuris.

The causes for so extensive a parasitism are : defaecation anywhere in gardens, fields, cultivated lands ; prevalence of flies ; absence of protection of food ; failure to wear boots ; no control of carriers. Mention is made of remedial measures which should be undertaken by the estate officials and by the municipality. *H. H. S.*

Arenas (Rogelio), Herrada (Manuel) & Alvarez (José R.). Valor del examen parasitológico de las heces fecales frente al parasitismo intestinal verminoso revelado por la autopsia. [**Comparison between the Results of Examinations of Faeces and Post-mortem Findings.**]—*Rev. Parasit., Clin. y Lab.* Habana. 1936. May–June. Vol. 2. No. 3. pp. 461–467. English summary.

The subjects of these tests were 68 dogs killed by coal gas and the autopsies were performed from 20 minutes to 4 hours after death. The intestine from cardia to anus was removed, the rectal ampulla emptied by pressure and the contents (to correspond with normal faecal excretion) sent to the laboratory for examination in the usual way. The intestine was then laid open and examined for worms. The greatest discrepancies were found as regards Dipylidium. These were not found in any of the faecal examinations but were seen in 38 at the autopsy; by the latter Ancylostoma was seen in 42, in the faeces 43 times; Trichuris was found 3 times at autopsy, 4 times in faeces. With ascaris again there was marked difference—3 positive by faeces and 12 by autopsy. Once only was Diphyllobothrium found, but this was seen by both methods. In the article protocols give the findings of single infestations and the various combinations of them. [It would make an interesting study to carry out an analogous investigation in man, comparing the findings of ova during life or, as above, in the faeces soon after death and those revealed by autopsy.] *H. H. S.*

Hinman (E. Harold) & Baker (D. D.). Helminthological Survey of 1,315 Dogs from New Orleans with Special Reference to Age-Resistance.—*Jl. Trop. Med. & Hyg.* 1936. May 1. Vol. 39. No. 9. pp. 101–104. [20 refs.]

Southern Rhodesia. Annual Report of the Public Health Laboratory and Pasteur Institute [1935–36] [Blackie (W. K.), Director]. **The Pulmonary Manifestations of Bilharzia Disease** p. 9. **The Definitive Sites of** *B. haematobium* **in the Pelvis** p. 9.

The distribution of *Schistosoma haematobium* in the body is wide.

At autopsy the eggs were seen in nearly every case in the lungs, with typical miliary nodules and an X-ray picture which may be like that of pulmonary tuberculosis; and as to the pelvis with few eggs in the bladder there may be many in the Fallopian tubes, while the prostate, seminal vesicles and lower parts of the ureters are common sites.

C. L.

Day (H. B.) & Kenawy (M. R.). **A Case of Bilharzial Myelitis.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. July 31. Vol. 30. No. 2. pp. 223–224. With 1 fig.

The boy of ten died with complete paralysis of legs, anaesthesia of legs spreading to the trunk, incontinence of urine and faeces, bed sores, septic parotitis, pyaemia.

In the lumbar enlargement of the spinal cord were small necrotic areas. Under the microscope in the grey matter and just behind the anterior horns were ova of *S. haematobium* with granulation tissue

round them. Nerve cells near the ova were more or less degenerating with disappearing dendrons and dissolution of Nissl's granules and nuclei; the others were normal. There was no sign of infection in urinary or alimentary tracts and no worms were seen in the portal vein.

C. L.

Le Gac (P.), Espian (M.) & Rarivoson (A.). A propos d'un cas de bilharziose vésicale autochtone. [**An Indigenous Case of Urinary Schistosomiasis (in Madagascar).**]—*Bull. Soc. Path. Exot.* 1936. Apr. 1. Vol. 29. No. 4. pp. 431–432.

This, it is believed, is the first case of infection with *S. haematobium* in one who has never left Madagascar. *C. L.*

Cawston (F. Gordon). **The Lithium Salts of Antimony in Bilharzia Infection.**—*Prescriber.* 1936. July. Vol. 30. No. 7. pp. 233–234.

Anthiomaline has advantages in the treatment of schistosomiasis. Lithium antimony-thiomalate is very soluble, stable in the dark and below 80°C., contains 16 per cent. of antimony, its low toxicity may be due to sulphur in the molecule, it is clear when in solution, an advantage in being certain of entry into a vein in intravenous injection. In the ampoules supplied were 2 cc. of a 6 per cent. solution or 0·01 gm. of antimony. The doses are 0·5 cc. for the first to a child of 12, and 1·5 cc. for the others. The repeated adult dose should not be more than 4 cc., otherwise there will be salivation and retching. In this there is less than half the quantity of antimony than in the repeated dose of tartar emetic but dead eggs are sooner passed with anthiomaline. Four injections a week may be given till this condition is present, which is in about a month sooner than with tartar emetic, though the drug has had to be given for 6 or 7 weeks. A permanent cure has been got after less than 0·5 gm. of antimony. *C. L.*

Piéri (J.) & Sardon. A propos d'un cas de bilharziose vésicale traité par le 110 L (antimoniothiomalate de lithium). [**A Case of Bilharziasis treated with Lithium Antimoniothiomalate.**]—*Bull. Soc. Path. Exot.* 1936. May 13. Vol. 29. No. 5. pp. 508–513.

Treatment was successful in a child of 12 in doses of 1 and 2 cc. and thereafter of 3 cc. twice a week to a total of 30 cc. [Although only a single instance is reported, it is noted here in order to call attention to a new drug and its dosage.] *C. L.*

Bacchelli (Giulio). Sul primo caso di Bilharziosi intestinale da *Schi. mansoni* osservato nella Somalia Italiana. [**The First Case of *S. mansoni* Infestation recorded in Italian Somaliland.**]—*Arch. Ital. Sci. Med. Colon. e Parassit.* 1936. Apr. Vol. 17. No. 4. pp. 206–208.

An Arab had eggs of *S. mansoni* in the faeces, the first case seen in Italian Somaliland. He had come there from Yemen, Arabia, 6 months earlier, and the belief is that he had got his infection in Yemen [see this *Bulletin*, 1936, Vol. 33, pp. 11 and 14.]. *C. L.*

Satta (Ernesto). Identificazione del primo focolaio di "Bilharziosi intestinale" da *Sch. mansoni* nella Colonia Eritrea. [**A Focus of *S. mansoni* Infestation in Eritrea.**]—*Arch. Ital. Sci. Med. Colon. e Parassit.* 1936. Apr. Vol. 17. No. 4. pp. 193–205. With 5 figs. & 1 map. [22 refs.]

A case of Mansonian schistosomiasis infected in Eritrea.

An Italian, after a year in Eritrea, had a bath in a pool of the fast mountain stream, Dadda, got pruritus all over the body 10 hours later, and in 35 days fever and dysentery. There were eggs of *S. mansoni* in the faeces. In that stream *Planorbis boissyi asmarica* n. var. was present. Satta makes the statement that in his review of the literature of January, 1936 [this *Bulletin*, 1936, Vol. 33, pp. 1–15] Lane says that *S. mansoni* does not exist in Eritrea, Ethiopia, or French or English Somaliland [the words used were that from these places "No accounts have come to light," a very different thing], and makes the point that Girolami had collected cases in the Italian literature from Eritrea [see this *Bulletin*, 1936, Vol. 33, p. 553]. [The writer's intention to make clear the gaps which there are in the records has, then, had a quick and fruitful result.] *C. L.*

Farges. Sur quelques foyers de bilharzioses en Côte d'Ivoire. [**Foci of Schistosomiasis on the Ivory Coast.**]—*Ann. de Méd. et de Pharm. Colon.* 1936. Apr.–May–June. Vol. 34. No. 2. pp. 384–387.

The subject of the title of this paper is mainly of local importance only. The author finds, however, that anthiomaline is of equal value to tartar emetic, blood ceasing to be present in the urine after 0·2 gm.; it is remarkably well tolerated, and is easily given subcutaneously or intramuscularly, a point of importance in treating children and where injections are given by the unskilled. *C. L.*

Khalil (M.) & Azim (M. Abdel). **An Epidemic-like Increase of Mortality due to Intestinal Schistosomiasis in an Egyptian Village.**—*Jl. Egyptian Med. Assoc.* 1936. Aug. Vol. 19. No. 8. pp. 475–495. With 8 graphs, 3 maps & 2 figs.

In the village of Tanan, 25 km. to the north east of Cairo and with about 9,000 inhabitants, there took place in the autumn of 1934 and again of 1935 an unexplained rise in the death rate in men only. The symptoms were those of dysentery, debility, anaemia with haemoglobin down to 20 or 30, and eosinophilia. Malaria parasites were absent, microfilariae were present in 8 per cent. and enlarged spleen and liver in 5 per cent. When death took place it did so in 2 to about 24 months. In one girl who died there were many worms in subperitoneal nodules, such as are seen only in heavily infected animals. There were some worms in abdominal lymph glands and living eggs in liver and pancreas. From examination of urine and faeces of 396 inhabitants 55 per cent. showed eggs of *S. haematobium* (in males 86 per cent., in females 24 per cent.), while 59 per cent. showed those of *S. mansoni* (in males 59 per cent., in females 31 per cent.). The canal running through the village was piled with refuse and dead dogs. The mosque sewer opens into it and the mosque latrines are used very freely by those living near. *Physa acuta* and *Planorbis boissyi* are many and large in this canal. A scheme is on foot for a new irrigation canal to take the place of this

foul one and for the permanent closure of the mosque latrines. [The "Contents" of the journal are in error in attributing this paper to Khalil alone.] *C. L.*

TRIM (E. A.). **Schistosomal Cirrhosis and Splenomegaly in the Central Kavirondo District of Kenya Colony.**—*East African Med Jl.* 1936. Aug. Vol. 13. No. 5. pp. 130–141. With 3 figs. on 1 plate. [30 refs.]

Cirrhosis of liver, generally with splenomegaly, is present in more than 1 per cent. of inpatients in the native hospital, Kisumu; all of them lived where *S. mansoni* is prevalent and in half of them the eggs of the parasite were seen. The changes in blood, spleen and liver were like those of schistosomiasis, in fact they are cases of "Egyptian splenomegaly." *C. L.*

RODRIGUEZ-MOLINA (R.). Schistosomiasis mansoni, Cirrhosis of the Liver with Splenomegaly and Macrocytic Anemia.—*Bol. Asoc. Med. Puerto Rico.* 1936. June. Vol. 28. No. 6. pp. 119–120.

RODRIGUEZ-MOLINA (R.). **Anemia in Schistosomiasis Mansoni and its Treatment in Puerto Rico.**—*Bol. Asoc. Med. de Puerto Rico.* 1936. July. Vol. 28. No. 7. pp. 146–151.

A study of the blood in 20 cases of Mansonian schistosomiasis.

The blood was taken from a vein, 3 cc. being kept liquid by the addition of 6 mgm. of dry potassium oxalate, and 100 per cent. Hb. being taken as that present when there were 14·5 gm. in 100 cc. of blood. One case was seen in the first or toxaemic stage with red cells 5,460,000, Hb. 83, mean corpuscular Hb. 22 micro-micrograms (slightly below normal), mean corpuscular volume normal. In the second diarrhoeic stage 8 were seen; red cells 3,400,000 to 4,900,000, Hb. 55 to 110, the blood hypochromic microcytic in 5, normocytic in 3, the anaemia responded to iron and ammonium citrate 2 to 6 gm. daily, but neither fouadin nor liver extract had any influence. In the third visceral stage 11 were seen, in 6 the anaemia was macrocytic, in 3 microcytic, in 2 normocytic, the red cells were from 1,360,000 to 3,960,000, Hb. 30 to 100. In the 5 macrocytic cases there had been haemorrhage from the bowel and in one haematemesis, and it is a microcytic anaemia which goes with loss of blood, and this microcytic anaemia "seems to fit in with the clincal picture of cirrhosis of the liver, splenomegaly and anaemia encountered in Banti's syndrome. . . Can the macrocytic anaemia . . . in the late stages of schistosomiasis be considered as an indication of liver dysfunction?" In two cases splenectomy has been followed by marked clinical improvement. *C. L.*

ERRATUM.—Vol. 33. No. 7. RODRIGUEZ MOLINA & PONS summary pp. 555–556, 15th line from top of p. 556 *for* 'by the "specific antianaemic factor"' *read* 'of the "specific antianaemic factor"'.

GIOVANNOLA (Arnaldo). **Specific Action of Some Drugs on Experimental Infections of** *Schistosoma mansoni.*—*Amer. Jl. Hyg.* 1936. July. Vol. 24. No. 1. pp. 102–108.

Tartar emetic proved the best drug for the killing of schistosomes in experimental conditions.

The test animals were rabbits in 3 series, the last two having controls showing that 43 and 147 worms became parasites. They were infected either by dropping the same quantity of the same cercarial suspension on to the shaved abdomen or by being put with shaved abdomen in the same infective bath. They were then given a curative drug. After tartar emetic given intravenously in a 6 per cent. solution to a total of 75 mgm. in doses of 15 mgm. every other day, no living flukes were present; after acriflavine (2 per cent. in water freshly made and put into the stomach by tube in quantities of 40 mgm. on alternate days to a total of 200 mgm.) there were present "about 100," 36 and 73; after emetine hydrochloride injected intramuscularly in 4 per cent. solution on alternate days in doses of 10 mgm. (total 30 mgm.) or 5 mgm. (total 25 mgm.) the living worms numbered 12, "not done," and 6; after fouadin injected intramuscularly in 2 injections of 0·25 cc. and 7 of 0·5 cc. there were in the first series "about 200" and in the second 40 worms; after acriflavine by mouth (40 mgm. every other day and 200 mgm. in all) the surviving worms were "about 100," 36 and 73. "Fouadin did not show a specific action . . . Acriflavine was without use. . . . Emetin hydrochloride had some action . . . especially when given in smaller doses." None of the drugs prevented infection when given previous to it. *C. L.*

Cawston (F. Gordon). **The Duration of Treatment for Bilharziasis.**—*Jl. Trop. Med. & Hyg.* 1936. Sept. 15. Vol. 39. No. 18. pp. 212–213.

The comparatively large doses of tartar emetic given to children without ill effects should be got into a period of a month. *S. mansoni* infections need special care and attention. A patient given 40 cc. of anthiomaline has put on 7 lb. in 6 months and remains free from signs of infection. Boys of 8 and 10 have needed 30 cc. of the drug in a month to bring about cessation of passage of eggs. But in one such child a rise in eosinophils from 8 to 29 per cent. after treatment suggests care in pronouncing cure till these cells become normal in numbers. *C. L.*

Giovannola (Arnaldo). Unisexual Infection with *Schistosoma mansoni*.—*Jl. Parasitology*. 1936. June. Vol. 22. No. 3. pp. 289–290. With 1 fig.

Giovannola (Arnaldo). Comparative Action of Tartar Emetic and Fouadin on *Schistosoma mansoni* in vitro.—*Jl. Parasitology*. 1936. June. Vol. 22. No. 3. pp. 302–303.

Bindra (B. S.). The Treatment of Schistosomiasis. Extract from Thesis on "*Schistosoma japonicum*, and the Problem of Schistosomiasis in India."—*Jl. Trop. Med. & Hyg.* 1936. Aug. 1 & 15. Vol. 39. Nos. 15 & 16. pp. 175–179; 185–190. [88 refs.]

Kan (Huai-Chieh) & Kung (Jen-Chi). Incidence of Schistosomiasis Japonica in an Endemic Area in Chekiang.—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 449–456. With 3 figs. & 1 sketch map.

Spiridion (J. T.). Schistosomiasis Japonica: an Account of an Outbreak.—*Jl. Trop. Med. & Hyg.* 1936. July 15. Vol. 39. No. 14. pp. 161–164. [15 refs.]

Cawston (F. Gordon). Destruction of the Bilharzia Parasites of Man.—*Jl. Trop. Med. & Hyg.* 1936. July 1. Vol. 39. No. 13. pp. 150–152.

Watt (John Yin Chieh). **Study on the Bionomics of the Intermediate Host of** *Schistosoma japonicum* **in Kutang, Chekiang, 1934–35.**—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 434–441. With 1 map & 1 chart.

The bionomics of *Oncomelania* snails were studied for a full year. Their habitats varied with season, hibernation in earth taking place in winter. The highest percentage infection rates of 16,501 snails were—in March (3·18) and September (3·4), the mean for all months being 1·28. Breeding takes place about May and September. *C. L.*

Edgar (W. H.). **Report on a Case of Epilepsy due to Infection by the** *Schistosomum japonicum.*—*Jl. Roy. Nav. Med. Serv.* 1936. Apr. Vol. 22. No. 2. pp. 150–153.

Epileptiform convulsions, preceded by closing in of the right side of the field of vision, some worsening of sight, and an occasional fumbling for the right word, a paracentral scotoma in the right inferior temporal quadrant, and some aching on the left parietal bone led to operations which displayed under this bone a sharply outlined spherical yellowish tumour an inch in diameter which was pronounced " to consist of the ova of *S. japonicum.*" A month later the condition was much improved. *C. L.*

Kan (Huai-Chieh). **Intracutaneous Test with** *Schistosoma japonicum* **Antigen. (A Preliminary Report.)**—*Chinese Med. Jl.* 1936. Feb. Supp. No. 1. pp. 387–393.

" *Preparation of Antigen.*—The antigens which were used were prepared as follows : Adult *Schistosoma japonicum* were obtained from experimentally infected rabbits. *Taenia crassicollis* or *Clonorchis sinensis*, which were used as the control antigens, were obtained from infected animals. The helminths were washed with saline and dried in a desiccator. The dry helminths were then powdered and for one week extracted in a shaking machine with 1/10 normal sodium carbonate solution containing 1 per cent. phenol, using 100 parts of phenolized sodium carbonate solution to 1 part of the dry powder. The suspensions were separated by being centrifuged at a speed of 3,000 r.p.m. for 30 min. One part of the supernatant fluid was then diluted with 31 parts of normal saline, thus giving a concentration of 1 part of dried powder in 3,200 parts of diluents. This has been found to be satisfactory.

" The phenolized saline for control was prepared by adding ½ cc. of phenol to 100 cc. of normal saline."

The following is the author's summary of tests made with this antigen :—

" (1) Intracutaneous injections of *Schistosoma japonicum* antigen were given to 624 persons, using *Taenia* or *Clonorchis* antigen and phenolized saline as the controls.

" (2) Of the 101 females tested, 26 were positive to the test.

" (3) Of the 523 males tested, 397 were positive to the test.

" (4) Of the 423 persons in the positive group, 89·4 per cent. resided in the endemic regions.

" (5) In 48·6 per cent. of the cases in the positive group the result of this test agreed with the findings of the physical examination.

" (6) In 60·4 per cent. of the cases in the positive group results of this test agreed with those of the faecal examinations." *C. L.*

i. KHALIL (M.). **Individual Variation in the Excretion of Drugs as an Important Factor in their Therapeutic Results. A Practical Method for detecting the Schistosomiasis Cases with So-called Idiosyncrasy to Antimony to avoid Fatalities and Complication.**—*Jl. Egyptian Med. Assoc.* 1936. June. Vol. 19. No. 6. pp. 285–305. (No. 8. pp. 502–510. Discussion.)

ii. ——. **Excretion of Drugs : its Influence on Therapeutic Results with Special Reference to the Antimony Treatment of Schistosomiasis.**—*Lancet.* 1936. July 18. p. 132.

i. Persons vary much in their rate of excretion of antimony (and arsenic). Those whose excretion is quick are resistant to treatment with the drug and have to be given a series of courses. Those whose excretion is slow are liable to poisoning whose end may be death. Those with a mean rate are cured normally.

Of 1,938 cases of schistosomiasis treated with fouadin 53 per cent. were cured after 9 injections, 21 after 11, 6·6 after 13, 3·4 after more than 13, and 16 gave up treatment. About 1 per cent. were intolerant of the drug and 0·2 per cent. died suddenly. " Most of these cases are never reported in Egypt." Relapses took place in 33 per cent. of cases. The hypothesis is put forward that " the facts known can be all explained on the basis that individuals vary in their power of excretion of antimony from their bodies." Fouadin is a solution of a compound of antimony and pyrocatechin. The test used is for pyrocatechin in the urine and is this. To 5 cc. of clear urine add 5 cc. of a fresh 0·05 per cent. $FeCl_3$ solution. After shaking add 1 cc. of a strong ammonia solution and shake. The colour, at first green, changes to violet and then red with the strong alkali. The reaction can be brought about with ferrous salts and so is not due to catechol. The colour begins to be seen in some persons 15 minutes after an intramuscular injection of fouadin, in others there is none even after 24 hours, in the normal case it is seen after 3 hours. " The delay in excretion corresponds to the kidney efficiency as tested by the urea concentration test and blood urea. Cases that excrete fouadin quickly need more injections to get cured. . . . The intensity of the reaction corresponds roughly to the amount of antimony excreted judging by the stain on the copper foil of the Reinsch's test." As to treatment " The correct way is to calculate the dose according to the amount retained in the body for a reasonable time. This can only be done by quantitative estimation of the amount excreted within a few hours and this is the aim we are at present trying to attain."

As to the Egyptian problem : at an estimate there are 10 million cases of schistosomiasis, about one million take treatment every year, 2,000 of them are killed by antimony. This " fouadin tolerance test " may be used as a kidney efficiency test.

Pages 502–510 contain a discussion by 9 persons on the paper just summarized. GUINENA asked whether there were cases of catechol retention with ordinary urinary findings ; Khalil in answer said that there were such cases with apparently normal kidneys ; " this agrees however with the result of the urea concentration tests when done on

the same cases." ABDEL SHAFIE MOHAMED spoke of two cases noted by Khalil with renal tumours and slow excretion of the drug ; since these tumours are rarely bilateral and the good kidney is normal or hypertrophied there should not be retention of the drug. Again, to settle the question of excretion, parallel tests should be made on blood and urine. In reply Khalil said that in them there was a high blood urea and " disturbance of the urea concentration test " ; no tests had been made for antimony in the blood.

MAKAR said that in chronic infection of the bladder, specially with carcinoma, there were living ova. " It is well known that the kidneys of such patients are usually in an advanced stage of disease " nor could he agree to use a dangerous test for kidney function when there were safe ones to hand, for " cases with dormant uraemia are waiting for such indiscretion to have their fate sealed. Besides the drug which in scientific hands may be safely used as germicidal, is apt in the hands of less conscientious persons to become homicidal." Khalil replied that there was nothing to show that cases of bilharzial cancer were resistant to treatment ; that the test depends not on antimony but on pyrocatechin disulphonate of sodium and that deaths from the drug take place only after 6 or more injections.

I. R. FAHMY pointed out that the excretion of antimony continues after that of catechol ends and that so long as we are without knowledge of the rate of decomposition of catechol or fouadin in the body it is better to use a direct test for antimony than an indirect one for catechol. ALI HASSAN, too, pointed out that antimony may be found in the urine without the organic fouadin radicle, and questioned the attribution of death to the kidneys in a child mentioned in whom no lesions worth noting were found in them. To them Khalil replied that the test must be a simple one as this is, and that the results of the fouadin tolerance test and that for antimony corresponded " exactly." " The rate of antimony excretion, however, goes on much more slowly than that of the pyrocatechol radicle."

AMIN SABRI pointed out that, whatever might be said of antimony, cases have died from a first or second injection of 0·15 gm. of salvarsan or neosalvarsan. Khalil said in reply that the drug must have been impure.

SMIRK pointed out that the tests advised were not quantitative as they should be.

MAHMOND BEY MAHER dwelt on the medico-legal aspects of these deaths.

ii. Covers a part of the first paper. *C. L.*

MOULINARD (M.) Traitement de la bilharziose par l'anthiomaline. Résultats comparés à ceux obtenus avec l'émétine et l'émétique. [**Anthiomaline compared with Emetine and Tartar Emetic in the Treatment of Schistosomiasis.**]—*Ann. de Méd. et de Pharm. Colon.* 1936. Apr.–May–June. Vol. 34. No. 2. pp. 352–371.

Anthiomaline is a better drug against schistosomes than emetine or tartar emetic.

Eight cases were treated with each drug. The daily and total doses given to boys of 12 to 14 were—for emetine 0·04 and 0·6 gm., for tartar emetic 0·08 and 1·2, and for anthiomaline 0·12 (0·06 on the first day) and 1·38 gm. The mean cures per cent. at the end of treatment and some months later were, as judged by eggs—for emetine 62·5 and 12·5,

for tartar emetic 75 and 25·0, and for anthiomaline 100 and 85·7. To the last there was seen no contraindication, the injections were not painful, there was no local or general reaction, and no evidence of renal damage. *C. L.*

Richet (Pierre). Auto-observation d'un cas de bilharziose vésicale et de son traitement par différents composés stibies. [**Antimony Compounds in the Treatment of Urinary Schistosomiasis.**]—*Ann. de Méd. et de Pharm. Colon.* 1936. Apr.–May–June. Vol. 34. No. 2. pp. 372–383.

Anthiomaline has given the author the best results in his own person. A chronic infection needs chronic treatment. Malefern, bismuth, emetine and the arsenicals are absolutely useless. Antimony is not strictly a specific, the most active antimonials are fouadin and anthiomaline. *C. L.*

Robertson (R. C.). **The Breeding Conditions of** *Oncomelania hupensis* **(Rissoidae : Triculinae).**—*Lingnan Sci. Jl.* (continuation of *Lingnaam Agric. Rev.*). 1936. Vol. 15. No. 1. pp. 55–56. With 3 plates.

" It appears to us from the evidence collected so far that schistosomiasis is not likely to be contracted after the end of September and that mollusks are relatively free from infection until the end of the breeding season in the following spring." *C. L.*

Cawston (F. G.). The Local Application of New Remedies for Bilharziasis. [Correspondence.]—*South African Med. Jl.* 1936. Apr. 11. Vol. 10. No. 7. pp. 277–278.

YAWS AND SYPHILIS.

SAUNDERS (George M.), KUMM (Henry W.) & RERRIE (James I.). **The Relationship of Certain Environmental Factors to the Distribution of Yaws in Jamaica.**—*Amer. Jl. Hyg.* 1936. May. Vol. 23. No. 3. pp. 558–579. With 6 maps. [14 refs.]

In this article are detailed the results of studies and observations (conducted with the support and under the auspices of the International Health Division of the Rockefeller Foundation and the Government of Jamaica) upon the present distribution of yaws in Jamaica as it is related to the environmental factors of rainfall, geology, topography, the presence of Hippelates flies, etc.

Though the disease has the opportunity of spreading to every part of the island as infective cases are constantly being introduced into all areas, yaws is localized in Jamaica to certain sharply delimited areas, thus suggesting that certain factors limit the spread of the disease and others favour its propagation. The results to date cannot be better expressed than in the authors' own words :—

" Rainfall seems to have an important influence on the prevalence of yaws, although we cannot be certain whether it affects the disease directly, causing more infectious lesions to appear, or whether it is important indirectly in that it favors a dense vegetation. This in turn might affect distribution by providing suitable breeding places for insect vectors, by increasing the likelihood of injuries to the lower legs and feet, where the primary yaws lesion nearly always appears, or by maintaining the viability of the infecting organism outside the body. The underlying geological formation seems to be an important factor, for wherever we find porous white limestone there is little or no yaws. This may be because the ground dries very rapidly in such places and because there is usually a poor surface soil and scant vegetation. On the other hand, in most places where there is a relatively impervious formation, with productive soil, ample precipitation, and a jungle type of flora, yaws is common. Altitude seems to have little direct effect, for we find yaws at all elevations where there is a country peasant type of population. It may, however, have an indirect influence, because precipitation tends to be greater over hills than over low-lying plains. The sanitary status of the country peasant is essentially the same throughout the island ; but in districts where yaws is found, the disease is commonest among the lowest social orders. In fact, it may be said categorically from our experience that yaws almost never occurs among the upper classes and that a low standard of living is essential to the prevalence of the disease. Villages and towns always show less yaws than the surrounding country. This may be due to the presence of a slightly higher social order in the town or to a scantier vegetation. Wherever there is much yaws, *H. pallipes* flies are found in abundance ; and as a rule the opposite is true, although there are places where these flies are found in great numbers and where the disease is absent. Also, in comparing the incidence of yaws with the fly catch in adjoining urban and rural districts we find that, although the disease is much commoner in rural parts, the fly catch is essentially the same as in the urban parts. We cannot yet decide whether the distribution of *H. pallipes* in Jamaica determines the distribution of yaws, or whether both are affected by the same environmental factors, causing their distribution to coincide."

H. S. Stannus.

Kumm (Henry W.). **The Jamaican Species of *Hippelates* and *Oscinella* (Diptera, Chloropidae).**—*Bull. Entom. Res.* 1936. July. Vol. 27. Pt. 2. pp. 307–329. With 6 figs. & 1 plate. [16 refs.]

The author has focussed attention on these flies because they are the insects which were found in greatest abundance feeding on the ulcers of yaws, and are possibly important as vectors. There are fifteen species of Chloropidae in Jamaica, of which *Hippelates pallipes* Loew., is the most widely spread and abundant. The distribution of all the species in the island is described in detail and a key with clear explanatory diagrams for identification is given. *V. B. Wigglesworth.*

Kumm (Henry W.). The Natural Infection of *Hippelates pallipes* Loew with the Spirochaetes of Yaws.—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1935. Nov. 25. Vol. 29. No. 3. pp. 265–272. With 1 chart & 3 figs. on 2 plates.

Kumm (Henry W.) & Turner (Thomas B.). **The Transmission of Yaws from Man to Rabbits by an Insect Vector, *Hippelates pallipes* Loew.**—*Amer. Jl. Trop. Med.* 1936. May. Vol. 16. No. 3. pp. 245–271. With 5 plates. [17 refs.]

A paper detailing the first successful transmissions of yaws to rabbits by means of Hippelates flies.

Treponemata are found in large numbers in the oesophagus of this fly after an infecting meal but they diminish rapidly so that none are found after 48 hours. There is no evidence of invasion of the salivary glands or proboscis, nor of cyclical development, nor of contamination of the legs. On the other hand it was found that this fly repeatedly regurgitates "vomit drops" containing treponemata during some hours after feeding. This appears to be the probable mode of transmission from patient to wound on the second subject. In the experimental work flies were allowed to feed alternately on a yaws lesion and then either on a granulating wound on the back of a rabbit or on a freshly scarified area on the scrotum. In other cases the inoculum consisted of the dissected out infected oesophageal diverticula.

It was found that the scrotum site was the more easily infected and it was also discovered that inoculations made in the winter, with removal of the animals from Kingston to New York for the incubation period were the more successful. [A very interesting point in experimental work.] *H. S. S.*

Chenoy (C. F.), Siddiqui (M. Ahsan) & Abraham (A. C.). **Investigation of Yaws (*Koya* Disease) in Warangal.**—*Indian Med. Gaz.* 1936. June. Vol. 71. No. 6. pp. 322–326. With 1 map.

Yaws has generally been considered to be very rare in India. The discovery of the disease in the Warangal district is therefore of considerable interest. So far in the state of Hyderabad it has been found confined to the jungle tract to the east (between the Mungeru river and the Kodavari). It is believed, however, by the authors that yaws may also exist on the other side of the Kodavari, in the East Godavari area and in the Chanda forest. [It would seem not improbable that yaws will be found much more widely distributed in India than has been supposed, when search is made among other primitive forest tribes.]

Persons of all ages were found to be suffering from the affection, the incidence being estimated at 5 per cent. Gonds and Dorras, primitive tribes of Dravidian origin, formed the bulk of the sick. They are spoken of as a semi-starved people wearing only a loin-cloth, who huddle together in their huts for warmth. They are, however, of cleanly habits in regard to their houses and persons. They believe that infection results from contact but blame the red ant as playing some part. They say that "Mothers suffering from 'Koya' (yaws) never have given birth to a child suffering from the disease."

[From the clinical description given there is little doubt that the disease is framboesia though the descriptive terms used would appear to be sometimes rather loose.]

Sabre-tibia is not an uncommon affection, gangosa occurs and mention is made of a case with ulceration of the forehead and necrosis of the frontal bone. No cases exhibiting lesions of the mucous membranes were seen and no mention is made of J.A.N. or goundou. *H. S. S.*

HACKETT (Cecil J.). **A Critical Survey of Some References to Syphilis and Yaws among the Australian Aborigines.**—*Med. Jl. Australia.* 1936. May 30. 23rd Year. Vol. 1. No. 22. pp. 733–745. [51 refs.]

In this paper the author gives a critical review of all or nearly all that has been written upon the occurrence of syphilis and yaws in the Australian aborigines. It is for this reason impossible to summarize and Dr. Hackett's conclusions only will be given. He suggests that the references made by the older writers to venereal disease probably all refer to yaws and not syphilis, granuloma pudendi being excluded. He believes that when Australia was discovered by Europeans yaws was prevalent in all communities and that this disease persists among them so long as they are not divorced from their bush fashion of life. Yaws is endemic in the Northern Territory and in Central Australia in areas where syphilis is unknown. *H. S. S.*

HACKETT (Cecil J.). **Boomerang Legs and Yaws in Australian Aborigines.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1936. July 31. Vol. 30. No. 2. pp. 137–143. With 1 map & 4 figs. on 2 plates. [19 refs.]

A very interesting paper dealing with the condition, which in Australia has been referred to as boomerang leg, its relation to yaws and their distribution in that continent.

The author has shown that among the aborigines of the centre of Australia there is a widespread disease known to them as *irkintja* which he has identified as yaws. The disease is contracted in infancy or early childhood, lasts a certain time and then clears up. After a quiescent period of some years pains in the shins develop and the tibiae become bent, with the production of boomerang leg, practically always before puberty. Actual full blown cases of yaws were not seen but the association of the scars of the disease with the characteristic tibial deformation was a very close one. These scars are identical with those produced by yaws in the Northern Territories where yaws is well recognized and in the areas investigated syphilis is unknown.

The evolution of the pathological process is traced and Dr. Hackett makes references to some other bony changes which he thinks may be due to yaws including changes in the skull.

The high incidence of boomerang leg was remarkable and in the discussion some speakers suggested that a second factor might be involved. Diseased bones had been collected from widely separated areas in Australia and arguing from this Dr. Hackett suggested that in the past yaws had been widely diffused over Australia and had occurred among native groups living in non-tropical climates. In discussion it was pointed out that tropical and non-tropical climates must be differentiated from the climates within and without the geographical tropics. *H. S. S.*

Lambert (S. M.). A Yaws Campaign and an Epidemic of Poliomyelitis in Western Samoa.—*Jl. Trop. Med. & Hyg.* 1936. Feb. 15. Vol. 39. No. 4. pp. 41–46.

Alfredo Fahsen (V.). Contribución al estudio del pian en Guatemala. [**Yaws in Guatemala.**] [Thesis presented to the Faculty of Medical Sciences, National University, Guatemala 1933, Feb.]—44 pp. With 3 figs. 1935. Guatemala : Tipograf. Sánchez & de Guise.

This is a graduation thesis and has the usual characters of works of that kind. The present might have been applied to the disease in any locality for it contains no figures regarding local prevalence, in fact the only reference to Guatemala may be summed up in the conclusion that "Yaws exists in Guatemala, is endemic in a certain district, Chocola in the Suchitepéquez department, and has probably been so for many years." The description is divided into sections dealing with the geographical distribution, the spirochaete, its staining and cultivation, experimental inoculation in monkeys, the pathological anatomy, symptoms, prognosis and treatment. One looks in vain for a reasoned discussion of the unicist and dualist sides of the question of yaws and syphilis. The time-honoured list of text-book differences is given, and a repetition of the statements that yaws does not affect mucosa or viscera, etc.—each a mere *petitio principii*. The one difference which we have not seen stressed before is that in the discharge from yaws lesions the spirochaetes are numerous, 6–8 per field, which is not the case with syphilis—an unsafe guide [the micro-photograph reproduced shows three]. There are notes of 6 cases and an appended bibliography showing the sources from which most of the statements have been culled.

H. H. S.

van der Schaar (P. J.). De liquor cerebrospinalis bij lijders aan framboesia tropica. [**The Cerebrospinal Fluid in Tropical Yaws.**] —*Geneesk. Tijdschr. v. Nederl.-Indië.* 1936. Mar. 31. Vol. 76. No. 13. pp. 784–804. [16 refs.]

A total of 129 cases was subjected to examination and care was taken to avoid the inclusion of cases of syphilis, by attention to the history of the patient and any venereal lesions which he might carry. In 90 cases the cerebrospinal fluid showed no evidence of deviation from normal, while in 28 cases (24 per cent.) there was some indication of change. Positive Wassermann and Sachs-Georgi reactions can occur. More frequent was the finding of a positive protein reaction. Pleocytosis

was also included as a definite change in the condition of the cerebro-spinal fluid. One of the most noteworthy results of the research was that no change could ever be found in the fluid during the secondary or papular stage of the disease. This is significantly the stage where no doubt can exist as to the diagnosis between yaws and syphilis.

W. F. Harvey.

CARMAN (John A.). **A Case of Simultaneous Infection with Yaws and Primary Syphilis.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1935. Nov. 25. Vol. 29. No. 3. pp. 261–263.

The title of this article might suggest that the patient contracted both yaws and syphilis at the same time but in truth it is the report of the case of an East African native who developed yaws in April in his native village, contracted syphilis after intercourse with a native prostitute in Nairobi in the beginning of December. The evidence of yaws was a typical generalized granulomatous eruption ; the evidence of syphilis was an indurated spirochaete-positive penile ulcer with generalized enlargement of lymphatic glands.

Dr. J. H. SEQUEIRA who saw the case stated that while on the one hand the chancre was syphilitic without possible doubt, the granulomatous eruption was typical of yaws and never reproduced in syphilis.

H. S. S.

WILLIAMS (Herbert U.). **Pathology of Yaws. Especially the Relation of Yaws to Syphilis.**—*Arch. Pathology.* 1935. Oct. Vol. 20. No. 4. pp. 596–630. With 12 figs. [80 refs.]

This article forms a very readable general review of yaws from the pathological aspect but contains no original contribution to our knowledge of the disease nor any facts not already known to readers of this *Bulletin*, while the author's conclusions differ hardly at all from those expressed from time to time by the reviewer. *H. S. S.*

ASENCIO (Fernando). **Juxta Articular Nodules. Syphilitic Case Report and Review of Literature.**—*Bol. Asoc. Med. de Puerto Rico.* 1936. May. Vol. 28. No. 5. pp. 99–102. With 4 figs. [14 refs.]

An account of a case of J.A.N. of 17 years standing in a 56-year-old Wassermann positive, male, white Porto Rican who had had syphilis at the age of 29. Nodules were present over each ulnar crest and a number were also present in both buttocks. Some account is given of the histo-pathological picture. This purports to be the first case of J.A.N. to be reported from Porto Rico. *H. S. S.*

BREIJER (H. B. G.), ELSBACH (E. M.) & HERMANS (E. H.). Nodosités juxta-articulaires. [**Juxta-articular Nodules.**]—*Nederl. Tijdschr. v. Geneesk.* 1936. Mar. 14. Vol. 80. No. 11. pp. 1100–1105. French summary (5 lines).

These juxta-articular nodules develop in the subcutaneous tissue and exhibit three zones microscopically—a fibrous peripheral zone, a middle zone with young connective tissue cells, infiltration cells and macrophages, and an innermost zone, which may be hyaline or necrotic. The authors examined 1,450 Indonesians of the personnel of the Rotterdam Lloyd, of whom about 60 per cent. were Madoerese and about 40

per cent. Javans, with the result that nodules were found in 2·2 per cent. of the former and only 0·7 of the latter. A therapeutic test with neosalvarsan or neosalvarsan and bismuth caused the nodules slowly but surely to disappear. Microscopic examination of excised nodules, however, did not settle satisfactorily whether these nodules were due to yaws, syphilis or to some allied affection and the examination of spirochaetes in sections was inconclusive. *W. F. Harvey.*

Nishiyama (K.) & Nagahama (M.). Ueber die Nodosités juxta-articulaires Jeanselme bei den Japanern mit besonderer Berücksichtigung des Röntgenbildes. [**Juxta-Articular Nodules in the Japanese.**]—*Japanese Jl. Dermat. & Urol.* 1936. Feb. Vol. 39. No. 2. [In Japanese pp. 152–165. With 15 figs. [15 refs.] German summary pp. 17–18.]

Two cases are recorded. The first, a girl of 9¾ years, living near Osaka, had suffered for 3 years from " rheumatic pains in the joints." Examination showed numerous swellings to the size of a walnut, the smallest being as large as a pea, situated on the extensor aspect of both wrists, along the forearms, on the dorsum of the right foot and the sole of the left. They were cystic and on puncture a greyish milky fluid was obtained in small amount, containing cholesterin, lime and phosphate. [Some at least of these, *e.g.*, those along the bones of the forearm would seem to be true rheumatic nodes.]

The second was a man of 52 years who had served in the Russo-Japanese war, but for three years had lived mainly near Osaka. He had suffered for 18 years from joint and tendon pains. The nodes in his case were largely about the joints—the carpal joints, elbow, knee and hip, and at the insertion of the left tendo Achillis. They varied in size from a bean to a " nut," though they felt cystic nothing was obtained on puncture. Wassermann and Meinicke reactions were negative in both cases. [From the general description and also from the situation, these nodules should more likely be regarded as rheumatic or some of them perhaps as ganglia, rather than J.A.N. as named in the title. There was no history of yaws and the tests for syphilis were negative.]

H. H. S.

Nicoletti (Ferdinando). Su di un'atipica elaiopatia provocata. Contributo alla conoscenza dell'autolesionismo in infortunistica. [**Lesion Artificially Produced by Injection of Oil.**]—*Riv. Sanitaria Siciliana.* 1935. June 1. Vol. 23. No. 11. pp. 819–822, 825–827. With 2 figs. [12 refs.] French summary (7 lines). [Summary appears also in *Bulletin of Hygiene.*]

The subject of this narrative was a bricklayer and mason, aged 26 years, who slipped and struck his right knee. The joint swelled and he was treated in hospital and kept under observation for 3 months. After another 3 months he again presented himself with four fairly distinct swellings about the right patella, the largest having a diameter of 5½ cm., with connexion with the overlying skin but slightly moveable on the subjacent tissues. Flexion of the knee was reduced to an angle of 125°, but was overcome by passive movement. The swellings were thought at first to be fibro-sarcomata or bursal inflammatory effusions as sequelae of the injury. The patient stated they had developed quite rapidly. Suspicion, however, was aroused and a needle inserted ;

an oily fluid was withdrawn which on examination, chemically and by the Wood light, proved to be vaseline.

The case is related here because of the similarity of the condition to juxta-articular nodules. *H. H. S.*

Plantilla (Fidel C.) & Mabalay (Epifanio). **Gangosa—a Clinical and Histologic Study of an Extensive Case.**—*Monthly Bull. Bureau of Health.* Manila. 1935. Oct. Vol. 15. No. 10. pp. 367–371. With 2 figs.

This case is published on account of the extensive nature of the lesion and at the same time to report the pathological findings. It is one, however, easily counterparted in any country where gangosa occurs; the pathological picture is normal for the condition. *H. S. S.*

Essed (W. F. R.). De oudste beschrijvingen van den rhinopharyngitis mutilans, tevens een historisch bewijs, dat deze aandoening van framboetischen en niet van luetischen aard is. [**The Oldest Descriptions of Rhinopharyngitis Mutilans and a Historical Proof that this is Yaws and not Syphilis.**]—Reprinted from *Feestbundel 1936 v.h. Geneesk. Tijdschr v. Nederl.-Indië.* pp. 563–578. With 4 figs. on 2 plates. [23 refs.]

The author regards the condition known as rhinopharyngitis mutilans or gangosa as a framboesia disease. Almost all of the article is occupied with setting forth his interesting views on the relations of framboesia (yaws) to syphilis. These centre mainly round the question whether the epidemic syphilis of the fifteenth century was, in reality, yaws and was a Spanish disease introduced by the early sailors from America. The author's work on the "Source of Syphilis" gives a greater wealth of detail than does this pamphlet. (See this *Bulletin*, 1934, Vol. 31, p. 221.) *W. F. Harvey.*

Ramos e Silva (J.). Ainhum e bouba. [**Ainhum and Yaws.**]—*Hospital.* Rio de Janeiro. 1936. Aug. Vol. 8. No. 8. pp. 833–840. With 3 figs. [10 refs.] French summary.

After a brief review of the literature on ainhum in Brazil the author describes the case of a man of 41 years with this condition affecting the fourth toe of the left foot. The patient stated that it had started 4 years previously, contemporaneously with certain lesions of the sole, which the author describes as identical with keratoderma punctatum seen in the Philippines and the Netherlands Indies and believed to be yaws. This patient had suffered from yaws at the age of 7 years. The author is of opinion that these are not merely coincident lesions, but suggests that they are connected aetiologically; his reasons being that yaws and ainhum often occur together in natives, and that in Brazil decline in one has been associated with decline in the other. [Without further evidence, these reasons are not very convincing.]

H. H. S.

Hudson (Ellis H.). **Hyperkeratoses and Depigmentations in Bejel.**—*Ann. Trop. Med. & Parasit.* 1936. Apr. 8. Vol. 30. No. 1. pp. 3–10. With 2 plates. [12 refs.]

The previous paper by this author on "Bejel" or endemic child syphilis of the middle Euphrates area was reviewed in this *Bulletin*,

1935, Vol. 32, p. 891. In the present communication he adds short notes with excellent photographs upon two conditions which up to date have been thought to be characteristic of yaws. The one is characterized by thickening and fissuring of the skin of the soles of the feet at points bearing the weight of the body and the heel: the second is a patchy depigmentation of the skin of the hands. Both have been previously noted by LACAPÈRE in Moroccan syphilis. As regards the first condition the author recalls BLACKLOCK's remark that probably if natives who contracted syphilis in childhood and went about barefooted were observed they would be found to exhibit this plantar lesion which had been held to be characteristic of yaws. In the Euphrates region the condition is very common, all are said to have had bejel and all have positive Kahn reactions. [The actual proof that the condition is due to Bejel is of course wanting and Hudson notes that a girl aged 6 had a similar fissuring dermatitis on the hands and arms but the observation is of very great interest.]

The second condition is equally interesting and as far as one can judge from the photographs the lesions on the hands certainly resemble very closely those which have been considered to be due to yaws. In neither case have they been proved to be due to Bejel and yaws respectively but again the observation is a most interesting one. The skin of the depigmented areas in Bejel is said to be otherwise normal and it is stated that occasionally the process involves the skin of the whole body producing a piebald condition. [As far as I am aware this more generalized form has never been recorded in yaws, whereas in yaws when the hands (and wrists) are affected the feet and ankles are also generally the seat of similar lesions. One is left in doubt whether the piebald condition is in fact of the same nature as that seen in the hands; non-syphilitic vitiligo or leucoderma is not rare in coloured races. It is to be hoped that further observations of as precise a character as possible, will be forthcoming.] *H. S. S.*

TURNER (Thomas B.). **The Resistance of Yaws and Syphilis Patients to Reinoculation with Yaws Spirochetes.**—*Amer. Jl. Hyg.* 1936. May. Vol. 23. No. 3. pp. 431–448. [17 refs.]

Experiments are recorded in which yaws and syphilis patients were inoculated intracutaneously with living yaws spirochaetes, which in most instances were shown to be virulent for man. The inoculum consisted of 0·1 to 0·2 cc. serum from a secondary yaws lesion shown to contain active *T. pertenue* by dark ground illumination; control inoculation consisted in the use of 0·1 per cent. formolized serum obtained in the same way; both injections made into the skin of the thigh or back. Subjects examined within 48 hours after inoculation, at weekly intervals during the incubation period and thereafter at 2-weekly intervals for at least 3 months. Development of a yaws lesion was considered a positive result, failure of a lesion to appear was considered indicative of immunity. In some cases confirmation of positive result undertaken by dark-ground examination. In rare instances pyogenic lesions were noted a few days after inoculation.

Eighteen patients with yaws, from 2 to 11 years of age, whose infection has been present for from 1 week to 7 years were inoculated from their own lesions. Results all negative except in a single case who had had yaws for 5 months and who presented a profuse eruption and bony lesions.

Six of this series were inoculated with heterologous spirochaetes at the same time. Two yielded positive results among those who were negative to autoinoculation. Conclusion—Resistance to autoinoculation develops early and persists while active yaws lesions are present.

Sixty-seven yaws patients were inoculated with heterologous strains of yaws spirochaetes with the following results :—

Duration of first infection in years	No. of patients	Results			
		Positive		Negative	
		Number	Per cent.	Number	Per cent.
Less than 1 ...	16	12	75·0	4	25·0
1 to 3 ...	14	10	71·5	4	28·5
3 to 8 ...	18	8	44·4	10	55·6
10 to 65 ...	19	3	15·8	16	84·2

Conclusion—Immunity develops to heterologous strains of spirochaetes : the immunity varies according to the duration of the natural first infection. Several years may elapse before patients become refractory to this second inoculation. It was also shown that among this same group of 26 patients with *active* lesions 57·7 per cent. were refractory ; of 23 to whom treatment had been given and in whom the disease had been latent for less than 1 year 17·4 only were immune ; of 18 in whom latency had obtained for 2 years or more 83·3 per cent. were immune, the latent stage having been preserved in some naturally, in others in consequence of treatment.

Conclusion—The development of immunity is retarded by treatment which interrupts the normal course of the disease.

The types of lesion produced by inoculation were of two kinds :— (*a*) proliferative : either a single large framboesiform papule always containing spirochaetes or a collection of folliculo-papules with scanty organisms ; (*b*) destructive : when no spirochaetes were found. There was no definite correlation between the type of lesion present in the naturally occurring disease and that produced by inoculation.

Conclusion—the current theory that destructive or gummatous-like lesions are due to a changed reactivity of the body tissues must be accepted with caution.

The incubation period on inoculation of 33 persons was from 13 to 84 days. In 27 the incubation period was under 6 weeks : in 11, inoculated with virus from a single case, the lesion appeared in 6 in the 3rd week, 3 in 4th week, 2 in 5th week.

Ten persons with latent syphilis in whom the diagnosis seems fairly assured were inoculated with *T. pertenue* shown to be virulent for man. The result was negative in all.

Three groups of patients comprising respectively (*a*) 10 with yaws of less than 3 years duration, (*b*) 10 with yaws of 10 years duration or more, and (*c*) 10 with latent syphilis were inoculated with the same testicular emulsion prepared from rabbits with yaws, adequate controls being used. Ten per cent. only of (*a*) were immune, 80 per cent. of (*b*), and 100 per cent. of (*c*).

It is suggested that successive crops of lesions which are commonly seen in yaws might be due to reinfections, a point of importance in the interpretation of the results of treatment.

It is pointed out that reliable information concerning cross immunity of the yaws patient to syphilis is not forthcoming and that even though many persons contract yaws in childhood their relatively susceptible period to syphilis may extend well beyond the adolescent years, so that caution must be observed in assigning *T. pertenue* as the aetiological agent in the case of syphilis-like lesions of the cardio-vascular and nervous systems. In the individual case, perhaps the most reliable method of arriving at a correct aetiological diagnosis is by recovery through rabbit inoculation of the one or the other treponema, with its characteristic disease picture, from the lymph nodes or other organs of the affected person. *H. S. S.*

Serra (G.). Le salicylate de bismuth dans la thérapeutique du pian. [**Bismuth Salicylate in Treatment of Yaws.**]—*Bull. Méd. du Katanga.* 1935. Vol. 12. No. 5. pp. 189, 191–7, 199–200.

An article dealing with the method of preparation, mode of employment and advantages of bismuth salicylate in the treatment of yaws. 100 gm. bismuth salicylate after complete drying by heat for two or three hours are triturated in a mortar with 1 litre of ground-nut oil added slowly till a fine emulsion results; this is then kept for 15–20 minutes on a water-bath with the water boiling, after which 10 gm. guaiacol is added. The dosage recommended is: 1 cc. per 10 kilo body weight intramuscularly into the buttock once a month for three to four months or 1 cc. per 15 kilo body weight once a week for five weeks. The injection is fairly painless, no toxic effects were seen, early lesions disappear quickly, it is cheap. Such a course of treatment, however, cannot be looked upon as curative. *H. S. S.*

Gotay (José B.), Costa Mandry (O.) & Payne (George C.). **The Intensive Method of Control applied to Yaws. A Preliminary Report.**—*Puerto Rico Jl. Public Health & Trop. Med.* 1935. Sept. Vol. 11. No. 1. pp. 91–101. With 4 figs. on 2 plates. [Spanish version pp. 102–113.]

A description of the results obtained by an intensive method of anti-yaws campaign.

In opening, reference is made to the history of the disease in the island of Porto Rico. Introduced with negro slaves during the second and third decade of the 16th century it remained a widespread infection until slave trading was discontinued in 1865, after which the disease rapidly declined and thereafter appeared only in small localized endemic foci scattered throughout the island.

In 1902–03 there was noted an epidemic among school children in the N.W. corner of the island which was mistaken for syphilis and treated apparently with success with mercury and potassium iodide but from 1903 to 1917 nothing further was reported. In 1917, however, cases were again noted and in 1931 an outbreak was signalled which forms the subject of this communication.

The intensive method of control consisted in :—(*a*) A reconnaissance by a sanitary inspector in order to delimit the affected zone. (*b*) A census of the total population of that zone with notification of all cases

and of those with suggestive histories. (*c*) The summoning of all those notified to a treatment centre. (*d*) Re-examination of the population at intervals.

The area involved was some 4 to 5 square miles in extent ; the total population numbered 1,091. Ninety-four cases of yaws and 34 persons with a suspicious history were found. Of the former 78·2 per cent. yielded positive W.R., of the latter 10·5 per cent. For comparison 517 members of an unselected population were examined serologically and 411 found to be negative.

Treatment consisted in series of injections of 0·6 gm. N.A.B. Re-examination of these patients three years later revealed that 56·9 per cent. still gave positive W.R., *i.e.*, over 50 per cent., notwithstanding the disappearance of clinical manifestations. The figures did not differ much among those who had had 12 or more treatments or among those who had had fewer treatments. *H. S. S.*

REVIEWS AND NOTICES.

Topley (W. W. C.) [M.A., M.D., M.Sc., F.R.C.P., F.R.S., Professor of Bacteriology & Immunology, University of London, Director of the Division of Bacteriology & Immunology, London School of Hygiene & Tropical Medicine] & Wilson (G. S.) [M.D., F.R.C.P., D.P.H., Professor of Bacteriology as Applied to Hygiene, University of London, London School of Hygiene & Tropical Medicine]. **The Principles of Bacteriology and Immunity.** 2nd Edition.—pp. xv+1645. With 276 figs. 1936. London: Edward Arnold & Co. [50*s*.] [Review appears also in *Bulletin of Hygiene*.]

The appearance of a new and extensively revised edition of Topley and Wilson's "Principles of Bacteriology and Immunity" may be hailed as something of an "event" in the world of bacteriology, pathology and hygiene. The book is sufficiently well known to professional bacteriologists that they will need no instigation to procure the new edition, but as it is yet a comparatively new work it may not be out of place to refer to some of the qualities which recommend it to a more extended circle of readers. It may be described (in its present form) as a one-volume system of bacteriology, and the authors have wisely realized that while some parts of the subject are best reduced to a synopsis, there are others which call for continuous and logical development. The very full and explicit descriptions of the various species of bacteria are particularly well given and obviously incorporate the results of much independent study. On the other hand, the chapters on immunity, which closely follow the treatment given in Professor Topley's "Outline of Immunity," are without doubt one of the best accounts of the subject ever written. As a pioneer educational work and as a synthesis of contemporary bacteriological thought it is difficult to overpraise the book but it would be a mistake to regard it as purely theoretical. From beginning to end the practical and in particular the hygienic aspects of the subject are most thoroughly treated, and we know of nowhere where the wheat of bacteriological and immunological applications has been so thoroughly sifted from the chaff. Its compendiousness, comprehensiveness and accuracy make this the almost ideal book for the shelves of the M.O.H. or indeed of any practitioner in medicine who is interested in one of the most progressive of the medical sciences. To the isolated worker, in the tropics or elsewhere, the book is a godsend for it is a first-class book of reference and at the same time provides opportunities for much fascinating continuous reading. We wish the authors of this excellent work long success and many future editions, but when we contemplate the immense pains which must go to maintain it at its present splendid standard we wonder if the wish is really a kind one. *C. C. Okell.*

Sociedad Argentina Patologia Regional del Norte. **Novena Reunión. . . . celebrada en Mendoza 1, 2, 3 y 4 de octubre de 1935. En homenaje a la memoria de Carlos Chagas. Primer Tomo. Enfermedad de Chagas.** [Report of Ninth Congress of Northern Argentine Pathological Society. Special Chagas Memorial Number.]—pp. vi+568. With numerous illustrations. 1936. Buenos Aires: Imprenta de la Universidad.

This volume of between 500 and 600 pages is a memorial work to Professor Carlos Chagas and consists of 42 papers by various authors,

all dealing with some aspect of American trypanosomiasis, Chagas's disease. Most of them, of course, cover the now familiar ground, recording clinical cases, finding of cases in fresh districts, animal hosts, pathogeny of the infection and so on, subjects which have received ample notice in the *Bulletin* during the past 20 years. One or two outstanding articles merit more than such passing reference here. First, one by Dr. Evandro CHAGAS on Experimental Infection of Man by the Trypanosome. He shows that infection by bite, if it occurs at all, must be very exceptional, in fact he is of opinion that the only mode worth attention is that of inoculation of the gut contents. Next Dr. Salvador MAZZA stresses once more the value of Romaña's sign, unilateral palpebral oedema, as diagnostic of the disease in its early stage, and the same author has studied the presence of natural infection of dogs and cats. Together with MIYARA, SALOMON and SANJURJO, MAZZA has investigated domestic animals and wild mammals in the environs of Mendoza as carriers of *T. cruzi*. Of the latter he names the following Edentata : *Chaetophractus vellerosus vellerosus*, *Chaetophractus vellerosus pannosus*, *Dasypus novemcinctus novemcinctus*, *Zoedyus pichy caurinus* ; of the Marsupialia : *Didelphis paraguayensis* and *Lutrealina crasicaudata paranalis* ; Cheiroptera (bats) : *Nyctinomus macrotis* and *Myotis levis* ; Canidae : *Pseudalopex culpaeus culpaeus* ; Mustelidae : *Grisonella ratellina*. Of these, the last two of the edentata, and those belonging to the last three groups—Cheiroptera, Canidae and Mustelidae—were all determined in 1935.

The work will not only prove a noteworthy memorial to Carlos CHAGAS, but is useful as a compendium for reference. *H. H. S.*

Printed under the authority of HIS MAJESTY'S STATIONERY OFFICE
By the South Essex Recorders Limited, High Road, Ilford.

(1831) Wt. P2/166 1875 12/36 S.E.R. Ltd. Gp. 353.

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. ,, Beriberi and Epidemic Dropsy.
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
C.Bu. ,, Climatic Bubo and Lympho-granuloma Inguinale.
Der. ,, Tropical Dermatology.
Diet. ,, Deficiency Diseases.
Dys. ,, Dysentery (Bacillary and Unclassed).
Fev. ,, Fevers.
Hel. ,, Helminthiasis.
Hist. ,, Historical.
H.S. ,, Heat Stroke.
K.A. ,, Kala Azar.
Lep. signifies Leprosy.
Lept. ,, Leptospirosis.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Oph. ,, Tropical Ophthalmology.
Pel. ,, Pellagra.
Pl. ,, Plague.
Rab. ,, Rabies.
R.B.F. ,, Rat-Bite Fever.
R.F. ,, Relapsing Fever and other Spirochaetoses.
Sn. ,, Venomous Snakes and Snake Venoms.
Sp. ,, Sprue.
S.S. ,, Sleeping Sickness.
Y.F. ,, Yellow Fever.
Y. & S. ,, Yaws and Syphilis.

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Acanfora, G., 502 (K.A.)
Acton, H. W. & Dey, N. C., 143 (Der.)
Adachi, J., with Kuo, Takahara & Saito, 627 (Misc.)
Adalja, K. V., (270) (Mal.)
Adams, A. R. D., 94, 105, 554 (Hel.), 462, 717 (Misc.), 650 (S.S.)
Adler, S., with Sergent, Ed., 499 (K.A.)
——, Theodor, O. & Schieber, H., 693 (R.F.)
Advier, M., with Mathis & Durieux, 340 (Y.F.)
Africa, C. M. & Garcia, E. Y., 91, 104 (Hel.)
——, with ——, 587 (Hel.)
——, —— & Layco, J., 131 (Hel.)
——, —— & de Leon, W., 90 (Hel.)
——, de Leon, W. & Garcia, E. Y., 560 *bis* (Hel.)
Ahern, D. M., with Dogra, 103 (Hel.)
Ahuja, M. L., 398 (Sn.)
——, with Taylor, 375 (Chl.), 387 *bis* (Sn.)
——, with —— & Gurkirpal Singh, 377 (Chl.)
di Aichelburg, U., 597 (Hel.)
Aida, T., (863) (Chl.)
Alayon, F., (371) (Pl.)
Albano, V., 502 (K.A.)
de Albuquerque, A. F. R., 601 (Lep.)
de Alda Calleja, M., 780 (Mal.)
Aleixo, A., 937 (Lep.)
Alexander, F. D. & Meleney, H. E., 528 (Am.)
Alfredo Fahsen, V., 963 (Y. & S.)
Alicata, J. E., 573, 591 (Hel.)
Allan, J., 476 (Misc.)
All-Union Institute of Experimental Medicine (Viem), 413 (B.R.)
Alvaré, L., with Kouri, Basnuevo & Lescano, 88, 558 (Hel.)
Alvarez, J. R., with Arenas & Herrada, 951 (Hel.)
Alves, W. D., 626 *ter* (Misc.)
Amalfitano, G., 27 (K.A.)
Ambialet, R., 263 (Mal.)
Amendola, F., 452 (Oph.)
de Amicis, A., 126 (Hel.)
Amy, A. C., 236 (Mal.)
Ancelot, A., with Launoy & Prieur, 203 (S.S.)
Anchezar, B., with Uriarte & Morales Villazon, 362, 370 (Pl.)
Anderson, A. S., with Simpson, 291 (Mal.)
Anderson, C., 18 (K.A.), 54 (R.F.)
Anderson, H. H. & Anderson, J. van D., 302 (Lep.)
——, with Hurwitz, 934 (Lep.)
——, with Reed, 540 (Am.)
Anderson, J. van D., with Anderson, 302 (Lep.)
——, with Villela & Castro, 940, (940) (Lep.)
Anderson, R. J., Crowder, J. A., Newman, M. S. & Stodola, F. H., 940 (Lep.)
Andrews, M. N., 16 (K.A.)
Anido, V., with Basnuevo, 549 (Hel.)
——, with Bolaños, Kouri & Basnuevo, 558 (Hel.)
Anigstein, L., 456 (Misc.)
Annales de l'Institut Pasteur, 325 (Rab.)
Annamalai, D. R., with Menon, 153 (Misc.)
——, with —— & Krishnaswami, 505 (K.A.)
Anning, C. C. P., 422 (Fev.)
Anton, H., with Hetsch, (710) (Dys.)
Antonino, L., 835 (Mal.)
Aoki, Y., 60 (Lept.)
Appelmans, M., 135 (Hel.)
——, with Van den Branden, 190 (S.S.)
——, with —— & Pottier, 202, 662 (S.S.)
Arantes, F. R., 314 (Lep.)

Arar, A., 118 (Hel.)
Arata, E. C., with Battaglia, 589 (Hel.)
Arbona, A., with Earle & Palacios, 837 (Mal.)
Archives de l'Institut Pasteur de Tunis, 326 (Rab.)
Arenas, R., Herrada, M. & Alvarez, J. R., 951 (Hel.)
——, with Kouri & Basnuevo, 587 (Hel.)
Arenas Martorell, R. & Herrada Llibre, M., 324 (Rab.)
Argerich, R., with Uriarte & Passalacqua, R., 361 (Pl.)
Arnaud, J., 256 (Mal.)
Arnett, J. H., with Stabler & Wenrich, 529 (Am.)
Arquivos da Escola Médico-Cirurgica de Nova Goa, 294 (Lep.)
Aschner, M., with Kligler & Levine, 427 (Fev.)
Ascione, G. & di Bello, G., 206 (S.S.)
Asencio, F., 964 (Y. & S.)
Atchley, F. O., 711 (Dys.)
Athavale, V. B., (315) (Lep.)
Audier, M., with Olmer, D. & Olmer, J., 910 (Fev.)
Augustine, D. L. & Drinker, C. K., 595 (Hel.)
——, with —— & Leigh, 129 (Hel.)
——, with Theiler, 592 (Hel.)
——, with —— & Spink, 123 (Hel.)
Auricchio, L. & Chieffi, A., 24 (K.A.)
Austin, C. J., 296, 599 (Lep.)
Austin, L. D. C., with Wolff, 635 (Misc.)
Austin, T. A. & Mayne, L. C., 527, 528 (Am.)
Autret, M., with Genevray, Gaschen & Dodero, 489 (Misc.)
de Azevedo, A. P., 109 (Hel.)
Azim, M. A., with Khalil, 92, 953 (Hel.)

B.

Bablet, J., 333 (Y.F.)
——, with Montel, Nguyen Ngoc Nhuan & Do van Hoanh, 944 (Lep.)
Bacchelli, G., 952 (Hel.)
Baer, J. G., with Joyeux, 86 (Hel.)
Baffet, O., with Nicolau, 322 (Rab.)
——, with —— & Mathis, 622 (Y.F.)
Bailly, J., with Remlinger, 316, 320, 325, 744, 745 (Rab.)
Baily, J. D., with Covell, (260) (Mal.)
——, with —— & Vidya Prasad, 226 (Mal.)
Baisas, F. E., (247) *bis*, (854) (Mal.)
——, with Russell, 487 (Misc.)
Baker, D. D., with Hinman, (951) (Hel.)
Baker, F. C., 285 (Mal.)
Baker, G. A. & Gibson, P. C., 392 (Sn.)
Balasheva, M. T., 23 (K.A.)
Balfour, M. C., 772 (Mal.)
Ballagi, S., 143 (Der.)
Baltazard, M., 917 (Fev.)
——, with Blanc, 918, 919 (Fev.)
——, with —— & Noury, 437, 924 (Fev.)
Balteanu, I. & Constantinesco, N., 920 *quat.*, 921 (Fev.)
Balteanu, J., with Ciuca & Constantinesco, 36, 925 *bis* (Fev.)
Bambarén, C. A., with Bernales, 40 (Fev.)
Bana, F. D., 618 (Y.F.)
Banerjee, D. N., 376 (Chl.)
—— & Datta, S. K., 378 (Chl.)
Banik, L. M., (711) (Dys.)
Bannerjee, K., with Pasricha & Lal, 924 (Fev.)
Barber, M. A., with Carr & Mandekos, 759 (Mal.)
—— & Rice, J. B., 258, 762 (Mal.)
Barbosa, A., 269, (789), (797) (Mal.)
Bariéty, M., with Troisier & Macrez, 352 (Lept.)
Barlovatz, A., 189 (S.S.)
Barlow, C. H., 93 (Hel.)
Barnard, C. C., 644 (B.R.)
Barnett, B., 391 (Sn.)
——, with Macfarlane, 396 (Sn.)
Barnett, L., 100 (Hel.)
Barros, J. M., 451 (Oph.)
Basaca, M., with Tubangui, Pasco & Del Rosario, 582 (Hel.)
Basnuevo, J. & Anido, V., 549 (Hel.)
Basnuevo, J. G., with Bolanos, Kouri & Anido, 558 (Hel.)
——, with Calvo Fonseca & Kouri, 108, 550 (Hel.)
——, with Kouri, 632 (Misc.)
——, with ——, Alvare & Lescano, 88, 558 (Hel.)
——, with —— & Arenas, 587 (Hel.)
——, with Kouri & Calvo Fonseca, 550 (Hel.)
Bassler, A., 705 (Am.)
Basso, R., with Mazza & Germinal, 207 (S.S.)
Basu, N. K., (315) (Lep.)
Battaglia, A. & Arata, E. C., 589 (Hel.)
Battaglia, M. I. & Uriarte, L., 361 (Pl.)
Beach, T. de V., 120, 588 (Hel.)
Bechelli, L. M., 314, 608 (Lep.)
——, with Valente, 313 (Lep.)
Bechuanaland Protectorate, 763 (Mal.)
Beckett, H. E., 629 (Misc.)
Beek, F. J. C., 629 (Misc.)
Beerens, J. & Cuypers, H., 389 (Sn.)
Belisario, J. C., 146 (Der.)
di Bello, G., with Ascione, 206 (S.S.)
di Benedetto, V., 504 (K.A.)
Benestad, G., (356) (R.B.F.)
Bengston, I. A. & Dyer, R. E., 441 (Fev.)
Benhamou, E., Faugère, R. & Choussat, F., 21 (K.A.)
——, & Fourès, R., 503 (K.A.)
——, & Gille, R., 505 (K.A.), 782 (Mal.)
Benitez, C., with Mazza & Janzi (676) (S.S.)
Bennett, S. C. J., 27 (K.A.)
Bensaude, A., with Cain & Cattan, 353 (Lept.)
Bensis, W. & Gouttas, A., 516 (Bl.)
Berberian, D. A., with Turner & Dennis, 101, (564) (Hel.)
Beretervide, J. J. & Masoch, T. J., 535 (Am.)
Berge, C., with Le Chuiton & Pennanéac'h, 439, 927 (Fev.)
——, with ——, Mondon, Pennanéac'h & Dubreuil, 438 (Fev.)
Berman, M., with Kligler, 667 (S.S.)
Bermingham, H. J., 337 (Y.F.)
Bernales, J. V. & Bambarén, C. A., 40 (Fev.)
Bernard, P. N., 860 (Chl.)
Bernard, R., with Giraud & Ciaudo, 503, 504, 509 (K.A.)
Berney, P., with Tisseuil, (301) (Lep.)
Berny, H., 936 (Lep.)
Berny, P., 612 (Lep.)
——, with Stévenel, 111 (Hel.), 301 (Lep.)

Berrebi, J., 501 (K.A.)
Berry, L. J., with Freeborn, 637 (Misc.)
Berthier, with Giraud, Ciaudo & Praliaud, 22 (K.A.)
Bertrand, C., with Montestruc, 127, 557 (Hel.)
Bertrand, Y., 451 (Oph.)
Berzi, A., (354) (Lept.)
Bessem, N., 443 (Fev.)
Besta, B. & Mariani, G., 305 (Lep.)
Bezemer, F., 474 (Misc.)
Bezerra, A., (844) (Mal.)
Bhaduri, B. N., 452 *bis* (Oph.)
Bhattacharjee, N. C., 776 (Mal.)
Bhattacharya, S. N., with Chopra, 405 (Diet.)
——, with —— & Sen, 519 (Bl.)
Bianchi, L., 501 (K.A.)
Bieling, R., 701 (Am.)
Bier, O. G., 305, 610 (Lep.)
Bilfinger, F., with Lépine, 508 (K.A.)
Bindra, B. S., (955) (Hel.)
Binford, C. H., 933 (Lep.)
Biraud, Y., 338 (Y.F.)
Bishopp, F. C., 241, (637) (Misc.)
——, & Smith, C. N., 485 (Misc.)
Bizard, M., 256 (Mal.)
Bjørn-Hansen, H., 519 (Bl.)
Blackie, W. K., 951 (Hel.)
Blacklock, D. B. & Southwell, T., 160 (B.R.)
Blair, A. W., 401 *bis* (Sn.)
Blanc, G., 925 (Fev.)
—— & Baltazard, M., 918, 919 (Fev.)
—— & Martin, L. A., 429 (Fev.)
—— & Noury, M., 699 (R.B.F.), 921, 924 (Fev.)
——, —— & Baltazard, M., 437, 924 (Fev.)
Blanchard, 456, 458 (Misc.)
Blanco, R. P., Capurro, F. G. & Dévé, F., 564 (Hel.)
Bodeau, 723 (Misc.)
Bodet, H., (874) (Pl.)
de Boer, E., 105, 574 (Hel.)
de Boer, H. S., 906 (Fev.)
van Bogaert, L., 657 (S.S.)
——, with Bourguignon & Van den Berghe, 668 (S.S.)
Bogliolo, L. & Greco, Z., 22 (K.A.)
——, with Liddo, 28 (K.A.)
Bogojawlenski, N. A., 838 (Mal.)
Bogomolow, I. L., with Kanarewskaja, 434 (Fev.)
Bohls, S. W., 422 (Fev.)
Bolaños, J. M., Kourí, P., Anido, V. & Basnuevo, J. G., 558 (Hel.)
Boldyr, E. D., with Petruschevsky, 98 (Hel.)
Bomskov, C., with Rominger, 70 (Sp.)
Boneau, E., with Laigret, 339 (Y.F.)
Bonebakker, A., 873 (Pl.)
Bonestell, A. E., with Kofoid & McNeil, 80 (Misc.)
Bonne, C., 113 (Hel.), 211 (S.S.), 338 (Y.F.)
—— & Stoker, W. J., 231 (Mal.)
Bonneau, E., with Durand, 615 (Y.F.)
Bonnefoi, A., with Martin & Rouessé, 620 (Y.F.)
Bonnel, with Sabrazès, Lannelongue & Dutrénit, 353 (Lept.)
Bonnin, H., 539 (Am.)
——, with Carles, 539 (Am.)
Boquet, P., with Césari, 384 (Sn.)
Bordjoški, M. & Milochevitch, S., 139 (Der.)
Borzone, A., (312) (Lep.)
Bose, P. N., with Krishnan & Ghosh, 521 (Bl.)
Boulin, with Labbé, Uhry & Ullman, 351 (Lept.)
Boulnois, J., 146 (Der.), 399 (Sn.)
Boulos, A., 587 (Hel.)
Bourgin, P., 264 (Mal.)
——, with Massias & Nguyen-van-Tan, 271 (Mal.)
—— & Nguyen-van-Tan, (228) (Mal.)
Bourguignon, 667 (S.S.)
Bourguignon, G. C., 203 (S.S.)
—— & Van den Berghe, L., with L. van Bogaert, 668 (S.S.)
Bouvier, G., 657 (S.S.)
——, with van Slype, 915 (Fev.)
Boyd, J. S. K., 417, 909 (Fev.)
Boyd, M. F., 227, 280 (Mal.)
—— & Kitchen, S. F., 842 (Mal.)
——, —— & Mulrennan, J. A., 843 (Mal.)
—— & Stratman-Thomas, W. K., 842 (Mal.)
——, —— & Kitchen, S. F., 240, 842 *bis* (Mal.)
——, —— & Muench, H., 779 (Mal.)
Boyé, R. & Rivierez, M., 145 (Der.)
Bozzelli, R., 318, 744 (Rab.)
Bradley, G. H. & McNeel, T. E., 486 (Misc.)
Braga, R., 312 (Lep.)
Brandt, R. & Horn, L., 268 (Mal.)
Brás de Sá, L. J., with de Mello, 259 (Mal.)
Brault, with Le Gall & Seyberlich, 874 (Pl.)
Breijer, H. B. G., Elsbach, E. M. & Hermans, E. H., 964 (Y. & S.)
van Breuseghem, R., 942, 944 (Lep.)
Briercliffe, R., 218 (Mal.)
——, Dalrymple-Champneys, W., Wigglesworth, V. B. & others, 679 (Mal.)
Brigham, G. D., 918 *bis* (Fev.)
British Empire Leprosy Relief Association, 598 (Lep.)
British Film Institute, 415 (B.R.)
British Medical Journal, 230, 279 (Mal.), 565 (Hel.)
Broc & Mami, A., 553 (Hel.)
Brodie, M., with Goodman, 42 (Fev.)
Brooke, G. E., 68 (Sp.)
Brown, A. A. F., 470 (Misc.)
Brown, A. F., 794 (Mal.)
Brown, H. W., with Lamson, 577 (Hel.)
Brown, J. A. K., 614 (Lep.)
Brown, P. W., 542 (Am.)
Browning, C. H., 195 (S.S.)
Bruck, A. J., 903 (Oph.)
Brug, S. L., (627) (Misc.)
Brumpt, E. & Lavier, G., 80 (Misc.)
Brunelli, P., 719 (Misc.)
Bruxelles-Médicale, (555) (Hel.)
Bruynoghe, R. & Jadin, J., 429 (Fev.)
Buchanan, G., 78 (Misc.)
de Buck, A., 227, 240 (Mal.)
——, with Kraan & Swellengrebel, 288 (Mal.)
—— & Swellengrebel, N. H., 241 *bis*, 242 (Mal.)
Budelmann, G. & Krauel, G., 518 (Bl.)
de Buen, S., (19) (K.A.), (284) (Mal.)
Bulletin de l'Académie de Médecine de Roumanie, 906 (Fev.)

Bulletin de l'Office International d'Hygiène Publique, 18 (K.A.), 333, 344 (Y.F.), 357 (Pl.)
Bulletin of the Ophthalmological Society of Egypt, 905 (Oph.)
Bunkall, J. D., with Cauchi & Sellers, 854 (Mal.)
Burgarella, M., with Debbasch & Calo, (839) (Mal.)
Burke-Gaffney, H. J. O'D., 640 (B.R.)
Burnet, E., 297 (Lep.)
Buschke, W., 904 (Oph.)
Busson, B., 747 (Rab.)
Bustamante, M., Varela, G. & Ríos Neri, F., 36 (Fev.)
Buxton, P. A., 278 (Mal.), 637 (Misc.), 873 (Pl.)

C.

Cáceres, R. & Izaguirre, A., 209 (S.S.)
Cain, A., Cattan, R. & Bensaude, A., 353 (Lept.)
Cairo, 453 (Oph.)
Calbairac, M. & Seyberlich, 369 (Pl.)
Calcutta School of Trop. Med. & Carmichael Hospital, 641, 642 (B.R.)
Calder, R. M. & Rigdon, R. H., 712 (Dys.)
Callot, J., 553 (Hel.)
Calo, A., with Debbasch & Burgarella, (839) (Mal.)
Calonne, R., 764 (Mal.)
Calvo Fonseca, R., Kouri, P. & Basnuevo, J. G., 108 (Hel.)
——, with —— & ——, 550 (Hel.)
Calwell, H. G., 695 (R.F.)
Cambrook, J. D., 392 (Sn.)
de Camelis, F., 839 (Mal.)
Cameron, G. R. & Karunaratne, W. A. E., 586 (Hel.)
Caminopetros, J., 23 (K.A.)
Campbell, T. C., with Defries, 744 (Rab.)
Camponovo, L. E., 713 (Dys.)
Campos, N. de S., 608 (Lep.)
Canal Feijóo, E. J., with Uriarte, 361 (Pl.)
Canciulesco, M. & Hirsch, R., (783) (Mal.)
Cano, M. & Pascual, A., (348) (R.F.)
Cantin, A. Y., with Kirk, 108 (Hel.)
Capurro, F. G., with Blanco & Dévé, 564 (Hel.)
Carbonell y Salazar, A. & Rodríguez y Remos, C., 576 (Hel.)
—— & Sanchez Santiago, B., 705 (Am.)
Carderera, M., with Montañes, 258 (Mal.)
Carles, J. & Bonnin, H., 539 (Am.)
Carman, J. A., 234 (Mal.), 410 (Diet.), 964 (Y. S.)
—— & Cormack, R. P., 793 (Mal.)
Caroli, J., 353 (Lept.)
Caronia, 499 (K.A.)
Carpano, M., 18 (K.A.)
Carr, H. P., Mandekos, A. & Barber, M. A., 759 (Mal.)
Carrión, A. L., 138, 143 (Der.)
—— & Emmons, C. W., 472 (Misc.)
Carrodus, A. L., 563 (Hel.)
Cartana, P., 24 (K.A.)
Cartia, G., 502 (K.A.)
Cartron, 296 (Lep.)
de Carvalho, J., 399 (Sn.)
de Carvalho, J. C., 313 (Lep.)
Casier, J., with Pergher, 423 (Fev.)
Cassuto, N., 268 (Mal.)
Castaneda, M. R., with Savoor & Zinsser, 436 (Fev.)
——, with Zinsser & Hager, 429 (Fev.)
Castellani, A., 140, 141 (Der.), 475 *bis* (Misc.), 541, 546 (Am.)
—— & Jacono, I., 139 (Der.)
—— & Standring, T., (473) (Misc.)
Castens, E., 136 (Hel.)
Castle, W. B., Rhoads, C. P., Lawson, H. A. & Payne, G. C., 66 (Sp.)
Castorina, G., with Timpano, 710 (Dys.)
Castro, A., with Villela, (611), (940) (Lep.)
——, with —— & Anderson, 940, (940) (Lep.)
Castronuovo, G., 73, (636) (Misc.)
Cataliotti, F., 564 (Hel.)
Catanei, A., 141 *ter* (Der.)
——, with Sergent, Edm., Sergent. Et. & Parrot, 757 (Mal.)
——, with Sergent, Et., 849 (Mal.)
Cattan, R., with Cain & Bensaude, 353 (Lept.)
Cauchi, J., Bunkall, J. D. & Sellers, W., 854 (Mal.)
Cawston, F. G., 97, (98), 556 *bis*, 557, 952, 955, (956), (959) (Hel.), 145 (Der.)
Ceccaldi, J., with Saleun, 342 (Y.F.)
Cervera, E., 400 (Sn.)
Césari, E. & Boquet, P., 384 (Sn.)
Ceylon, 214, 218 (Mal.)
Chagas, E., 500 (K.A.), 675, 676 (S.S.)
Chakraverti, C. R., 904 (Oph.)
Chan, M., 404 (Diet.)
Chand, S. D., with Hicks, 616 (Y.F.)
Chandler, A. C., 111, 120, (588) (Hel.), 567 (B.R.)
Chandra, S. N., with Ramsay & Lamprell, 843 (Mal.)
Chang, H. C., Ch'en, K. C. & Chou, S. K., 534 (Am.)
Chang, T. L., with Yen, 132 (Hel.)
Chang, W., with Lai & Li, 254 (Mal.)
Chang, Y., with Yang, Tsao & Chung, (865) (Chl.)
Chang, Y. H., Li, C. C. Young, M. P., 228 (Mal.)
Charamis, J., 903 (Oph.)
Chatterjee, H. N. Halder, M. N., 408 (Diet.)
Chatterjee, K., with De, 407, 408 (Diet.)
Chatterjee, N. C., (265) (Mal.)
Chatterji, S. N., with Muir, 299, 938 (Lep.)
Chaudhuri, R. N., 407 (Diet.)
——, with Chopra, 144 (Der.), 406 (Diet.), 721 (Misc.)
——, with —— & Panja, 145 (Der.)
Chauvin, L., 537 (Am.)
Chaves Belando, L. A., with Escomel, 808 (Misc.)
Cheissin, E., 491 (Misc.)
Chen, H. T., 92, 949 (Hel.)
Ch'en, K. C., with Chang & Chou, 534 (Am.)
Chen, W. Y., with Lai, 941 (Lep.)
Cheney, G., 446 (Fev.)
Ch'eng, Y. L. & K'ang, H. J., 564 (Hel.)
Chenoy, C. F., Siddiqui, M. A., & Abraham, A. C., 961 (Y. & S.)

Chesterman, C. C., 514 (Bl.)
Chieffi, A., 26 (K.A.)
——, with Auricchio, 24 (K.A.)
Chionetti, U., with Fusco, 630 (Misc.)
Chiyuto, S., 299, (604) (Lep.)
Chodukin, N. I., Petrov, V. P. & Kevorkov, N. P., 500 (K.A.)
——, Sofiev, M. S. & ——, 506 (K.A.)
Chodzko, W., 355 (R.B.F.)
Chopra, R. N., 407 (Diet.), 641, 642 (B.R.)
—— & Bhattacharya, S. N., 405 Diet..)
—— & Chaudhuri, R. N., 144 (Der.), 406 (Diet.), 721 (Misc.)
——, —— & Panja, D., 145 (Der.), 407 (Diet.)
—— & Chowhan, J. S., 397, (397) (Sn.)
——, —— & De, N. N., 389 (Sn.)
——, Gupta, J. C. & Roy, (A. C., 492 (Misc.)
——, —— & Sen, B., 795 Mal.)
——, with Krishnan & Mukherjee, 79 (Misc.)
—— & Mukherjee, S. N., 841 (Mal.)
—— & Roy, A. C., 273 (Mal.), 382 (Sn.)
——, Sen, B. & Bhattacharya, S. N., 519 (Bl.)
——, —— & Ganguli, S. K., 275 (Mal.)
——, —— & Sen, G., 545 (Am.)
Chorine, V., (230) (Mal.)
—— & Crougue, O., 940 (Lep.)
——, with Marchoux & Koechlin, 57 (R.F.), 307 (Lep.)
——, with Martin & Rouessé, 30 (K.A.)
—— & Prudhomme, R. O., 505 (K.A.)
Chou, S. K., with Chang & Ch'en, 534 (Am.)
Chonssat, F., with Benhamou & Faugère, 21 (K.A.)
Chowhan, J. S., with Chopra, 397, (397) (Sn.)
——, with —— & De, 389 (Sn.)
Christian, C. R., 50 (Fev.)
Christophers, R., 499 (K.A.)
Christophers, S. R., 213 (Mal.)
Chu, C. F., with Lee, 30 (K.A.)
Chu, H. J., with Lee, 96 (Hel.)
Chu, P. J., with Tao, Yu & Wang, 87 (Hel.)
Chun, J. W. H., with Wu Lien-Teh, Pollitzer & Wu, 726 (B.R.)
Chung, C. Y., with Yang, Tsao & Chang, (865) (Chl.)
Chung, H. L., 29 (K.A.)
—— & Lee, C. U., 103 (Hel.)
——, with ——, 97 (Hel.), 503 (K.A.)
Ciaudo, with Giraud, 23 (K.A.)
——, with —— & Bernard, 503, 504, 509 (K.A.)
——, with ——, Berthier & Praliaud, 22 (K.A.)
——, with Rouslacroix & Lumbroso, 506 (K.A.)
Cicchitto, A. M., 276 (Mal.), 713 (Dys.)
Ciferri, R. & Redaelli, P., 491 (Misc.)
Ciuca, M., Balteanu, I. & Constantinesco, N., 36, 925 *bis* (Fev.)
Claessen, G., 101 (Hel.)
Clark, E. & Graef, I., 96 (Hel.)
Clark, H. C., 280 (Mal.)
——, with Komp, 835 (Mal.)
Clements, F. W., 715 (Misc.), 839 (Mal.)
Cluver, E. H., 906 (Fev.)
Coatney, G. R., 238 (Mal.)
Cochrane, R. G., 298 (Lep.)
Coelho, J., 19 (K.A.)
Cogollo Duque, J., 443 (Fev.)
Colas-Belcour, J., 55 (R.F.)
——, with Roubaud, 653 (S.S.)
——, —— & Treillard, 288 (Mal.)
Cole, A. F., 699 (R.B.F.)
Coles, A. C., 691 (R.F.)
Collier, W. A. & Krause, M., 192 (S.S.)
Collignon, E., 244 *bis*, 263 (Mal.)
Colonial Development Fund (Malaria Research Scheme), 225 (Mal.)
Comaroff, R., with Kligler, 205 (S.S.)
Compagnini, G., (272) (Mal.)
Congo Belge, 645 (S.S.)
Connell, F. H., 527 (Am.)
Connery, J. E., with Goldwater, Steinberg & Most, 122 (Hel.)
Connor, F. P., 127 (Hel.)
Constantinesco, N., with Balteanu, 920 *quat.*, 921 (Fev.)
——, with Ciuca & Balteanu, 36, 925 *bis* (Fev.)
Cook, S. S., with Riley & Faust, 280 (Mal.)
Cooley, R. A., with Kohls, 693 (R.F.)
Copanaris, P., 18 (K.A.)
Copeland, A. J., (291) (Mal.)
Coria, N. A., with Glaser, 711 (Dys.)
Cormack, R. P., 465 (Misc.)
——, with Carman, 793 (Mal.)
Corman, A., 524 (Bl.)
Corradetti, A., 712 (Dys.), 806 (Misc.), 857 (Mal.)
Corsi, E. M., with Mazza, (678) (S.S.)
Corson, J. F., 649, 651, 667 (S.S.)
Cort, W. W., 554 (Hel.)
—— & Talbot, S. B., 554 (Hel.)
Costadoni, A., 267 (Mal.)
Costa Mandry, O., with Gotay & Payne, 969 (Y. & S.)
Costanzi, C., (921) (Fev.)
Cottrell, H. B., with Keller, Googe, Miller, Jr., & Harvey, 583 (Hel.)
Coutelen, F., 134, 563 (Hel.)
Covell, G., 226 (Mal.), 419, 910 (Fev.)
—— & Baily, J. D., (260) (Mal.)
——, —— & Vidya Prasad, 226 (Mal.)
—— & Mehta, D. R., 909 (Fev.)
Cowdry, E. V. & Heimburger, L. F., 301 (Lep.)
——, —— & Williams, P. S., 605 (Lep.)
Craig, C. F., 280 (Mal.), 526 (Am.)
Crainz, F., with Marchesi & Scapaticci, 18, 508 (K.A.)
Crimmins, M. L., with Peck & Erf, 395 (Sn.)
Cross, S. X., with Herrick, 669 (S.S.)
Crougue, O., with Chorine, 940 (Lep.)
Crowder, J. A., with Anderson, Newman & Stodola, 940 (Lep.)
Cruveilhier, L., with Lépine, 317 (Rab.)
——, Truche, C. & Viala, C., 746 (Rab.)
Cruz, W. O., 585 (Hel.)
Cuenca, H., (678) (S.S.)
Culbertson, J. T., 184 (S.S.), 485 (Misc.)
Cullen, T., 432 (Fev.)
Culpin, M., 72 (Misc.)
da Cunha, A. M., 428 (Fev.)
Cunningham, J. & Fraser, A. G. L., 54 (R.F.)
Curran, E. J., 431 (Fev.)
Cuypers, H., with Beerens, 389 (Sn.)

D.

Dale, W. C., 630 (Misc.)
Daleas, P., 228 (Mal.)
—— & Lavergne, J., 783 (Mal.)
Dalrymple-Champneys, W., with Briercliffe, Wigglesworth & others, 679 (Mal.)
Dang, J. M. L., 101 (Hel.)
Dang, M. G., Hemenway, R. V. & Lau, S., (777) (Mal.)
Dang-Hanh-Kiên, 524 (Bl.)
Danzel, L. A., (371) (Pl.)
Darder, E. & Meneu, A., 431 (Fev.)
Darré, H. & Mollaret, P., 619 (Y.F.)
Darriba, A. R., 595 (Hel.)
Das Gupta, B. M., with Knowles & Sen, 700 (R.B.F.)
Das Gupta, C. R., with Napier, 803 (Misc.)
Datta, S. K., with Banerjee, 378 (Chl.)
Daufresne, M., with Vinzent & Seguin, 692 (R.F.)
Davey, J. B., 654 (S.S.)
Davey, T. H., with Findlay, 615 (Y.F.)
——, with ——, Stefanopoulo & Mahaffy, 339 (Y.F.)
——, with Gordon, 514 (Bl.)
David, N. A., with Rosen & Martin, 545 (Am.)
Davies, W. M., 637 (Misc.)
Dawson, W. T., Gingrich, W. & Hollar, E. D., 272 (Mal.)
Day, H. B. & Kenawy, M. R., 951 (Hel.)
Day, L. F., 776 (Mal.)
De M. N. & Chatterjee, K., 407, 408 (Diet.)
De, N. N., with Chopra & Chowhan, 389 (Sn.)
Dearborn, F. E., 249 (Mal.)
Debbasch, G., Calo, A. & Burgarella, M., (839) (Mal.)
Decourt, P., 789 (Mal.)
——, Villain, G., Marini, C. & Dupoux, R., 797 (Mal.)
Defries, R. D. & Campbell, T. C., 744 (Rab).
Degotte, J., with Dubois, 303 (Lep.)
De la Barrera, J. M., 362 (Pl.)
—— & Riesel, M., 362 (Pl.)
Delanoë, E., 157 (Misc.), 449 (Oph.), 944 (Lep.)
Delbet, P., 597 (Hel.)
Delbove, P., with Ragiot, 38, 39 (Fev.)
—— with ——, Nguyen-van-Huong & Ho-thieu-Ngan, 354 (Lept.), 433 (Fev.)
Del Frade, A., with Kouri, 87 (Hel.)
Del Rosario, F., with King, 853 (Mal.)
——, with Tubangui, Basaca & Pasco, 582 (Hel.)
Del Toro Cano, F., 599 (Lep.)
Delville, J. P., 435 *ter* (Fev.)
Demidowa, L. W., 237 (Mal.)
Demjashev, M., with Fenjuk, 872 (Pl.)
Dendale, R., with Fabiani, 24 (K.A.), 535 (Am.)
Denning, G. M., with Wigmore, 354 (Lept.)
Dennis, E. W., with Turner & Berberian, 101, (564), (Hel.)
De Rivas, G. T., with Johnson, 675 (S.S.)
Dervillée, P., (587) (Hel.)
Deschiens, R., 532 (Am.)
——, with Dopter, 531 (Am.)
Des Essarts, J. Q., & Lefrou, G., 298 (Lep.)
—— with ——, 606, 936 (Lep.)
Despujols, B. P., 761 (Mal.)
Deutsch, V., with Levaditi, 57 (R.F.)
Dévé, F., with Blanco & Capurro, 564 (Hel.)
Devignat, R., (875) (Pl.)
Dew, H. R., 100 (Hel.)
Dey, N. C., with Acton, 143 (Der.)
Dhar, J., 77, 479 (Misc.)
Diaz Martin, L., 590 (Hel.)
Dibold, H., 712 (Dys.)
Diboll, C. G., with Faust, 280 (Mal.)
Dick, J. C., (565) (Hel.)
Dickson, R. M., 219 (Mal.)
Dierick, J. E. A. M., 806 (Misc.)
Diethelm, O. A., with Hughes & Tebbutt, 914 (Fev.)
Dimitrijević-Speth, V., 35 (Fev.)
Dimitriu, C. C. & Popovici, A., 265 (Mal.)
Dixon, D. S., with Walker, 638 (Misc.)
Dixon, H. B. F. & Smithers, D. W., 102 (Hel.)
Djohan, B., with de Langen, 409 (Diet.)
Dodd, M. C., with Gruhzit, Lindsay & Hendricks, 673 (S.S.)
Dodero, J., with Genevray, 321 (Rab.)
——, with ——, Gaschen & Autret, 489 (Misc.)
Dodge, C. W., 570 (B.R.)
Dogra, J. R. & Ahern, D. M., 103 (Hel.)
Donatien, A. & Lestoquard, F., 20 *bis* (K.A.), 46 (Fev.), 925 (Fev.)
——, with ——, 507 (K.A.)
Doorenbos, W., 375, 376 (Chl.)
Dopter, C. & Deschiens, R., 531 (Am.)
Dorolle, P. & Ngo-quang-Ly, 309 (Lep.)
——, ——, Huynh-van-Huy & Tran-van-Tam, 612 (Lep.)
Dostrovsky, A., 21 (K.A.)
Dostrowsky, A., 297 (Lep.)
Doull, J. A., Rodriguez, J. N., Guinto, R. & Plantilla, F. C., 931 (Lep.)
Do van Hoanh, with Montel, Bablet & Nguyen Ngoc Nhuan, 944 (Lep.)
——, with ——, Le-van-Phung, Nguyen-van-Khai, Tran-van-Hanh, Truong-van-Que & Nguyen-ngoc-Nhuan, 309 (Lep.)
Dreessen, W. C., with Williams, 344, 345 (Y.F.)
Drinker, C. K., with Augustine, 595 (Hel.)
——, —— & Leigh, O. C., 129 (Hel.)
Driollet, E., with Mazza, 211 (S.S.)
Drummond, R., (232) (Mal.)
Dubois, A., 298, 309, 942 (Lep.)
—— & Degotte, J., 303 (Lep.)
——, with Gavrilov, 610 (Lep.)
—— & Noël, G., 439 (Fev.)
Dubreuil, J., with Le Chuiton, Mondon, Berge & Pennanéac'h, 438 (Fev.)
Dufour, J., with Nattan-Larrier, 506 (K.A.)
Duggan, J. N. & Nanavati, B. P., 903 (Oph.)
Duke, H. L., 179, 196, 207, 645, 652, 657 (S.S.)
—— & Mellanby, K., 652 (S.S.)
Dunham, G. C., (600) (Lep.)
Dupoux, R., with Decourt, Villain & Marini, 797 (Mal.)

Dupoux, R., with Villain, 782 *bis* (Mal.)
Dupuy, 646 (S.S.)
Durand, R. & Bonneau, E., 615 (Y.F.)
——, with Laigret, 427 (Fev.)
Durieux, C., 915 (Fev.)
——, with Mathis & Advier, 340 (Y.F.)
Du Soleil, G., with Houssiau, 156 (Misc.)
Du Toit, P. J., 668 (S.S.), 749 (Rab.)
Dutrénit, J., with Sabrazès, Lannelongue & Bonnel, 353 (Lept.)
Duverger, M., with Fourrier & Genova, 500 (K.A.)
Dyckerhoff, H., 547 (Am.)
Dyer, R. E., with Bengston, 441 (Fev.)
Dyke, H. W., 763 (Mal.)
Dzen, M. Y. & Yu, H., 867 (Chl.)

E.

Earle, W. C., Palacios, L. D. & Arbona, A., 837 (Mal.)
Eddie, B., with Meyer, 871 (Pl.)
Edgar, W. H., 956 (Hel.)
Edney, J. M., with Nolf, 122 (Hel.)
Egan, C. H., 553 (Hel.)
Egypt, 452 (Oph.)
Ejército, A., (761) 774 (Mal.)
El-Biblawi, A., 544 (Am.)
Elisei, C., with Purcaro, 529 (Am.)
Ellison, F. O'B., 683 (Mal.)
El Salam, A. A., 95 (Hel.)
Elsbach, E. M., with Breijer & Hermans, 964 (Y. & S.)
Emmons, C. W., with Carrión, 472 (Misc.)
d'Empaire, J. R. H., (576) (Hel.)
Enneking, J. A. M. J., 69 (Sp.)
Epstein, E., 543 (Am.)
Epstein, G. W., & Silvers, I. L., 919 (Fev.)
——, —— & Exemplarskaya, E. V., 919 (Fev.)
Erber, B., 354 (Lept.)
——, with Mollaret, 350 *bis* (Lept.)
Erf, L. A., with Peck & Crimmins, 395 (Sn.)
Escalar, G., with Pecori, 757 (Mal.)
Escomel, E., 53 (Fev.), 631 (Misc.)
—— & Chaves Velando, L. A., 808 (Misc.)
Eskridge, L., with Hegner, 713 *bis* (Dys.)
Esménard, J., with Joyeux & Sédan, 135 (Hel.)
Espian, M., with Le Gac & Rarivoson, 952 (Hel.)
Espié, A., 87 (Hel.)
Essed, W. F. R., 966 (Y. & S.)
——, with Hidajat, 699 (R.B.F.)
Esser, P. H., 300, (605) (Lep.)
Esser, W., with Gessner, 492 (Misc.)
Eubanas, F., 602 (Lep.)
Evans, A. M., 848 (Mal.)
—— & Walker, G. R., (346) (Y.F.)
Evans, K. L., 625 (Misc.)
Exemplarskaya, E. V., with Epstein & Silvers, 919 (Fev.)

F.

Fabiani, G., 304 (Lep.)
—— & Dendale, R., 24 (K.A.), 535 (Am.)
Fabre, with Mercier & Soulage, 910 (Fev.)
Fadda, S., 257, (762) (Mal.)
Faddeeva, T., with Iwanowsky, 876 (Pl.)
—— & Tschernobaew, W., 875 (Pl.)
Fairley, N. H., 66 (Sp.)
—— & Mackie, F. P., 471 (Misc.)
Farges, 953 (Hel.)
Farinaud, E., (234) (Mal.)
Farinaud, M. E., 768 (Mal.)
Faugère, R., with Benhamou & Choussat, 21 (K.A.)
Faust, E. C., 94, 119 (Hel.)
—— & Diboll, C. G., 280 (Mal.)
—— & Headlee, W. H., (551) (Hel.)
—— & Martinez, W. H., 137 (Hel.)
——, with Riley & Cook, 280 (Mal.)
——, with Sayad & Johnson, 596 (Hel.)
—— & Swartzwelder, J. C., 547 (Am.)
Faworissowa, B., with Joukow-Werejnikow, 371 (Pl.)
——, with Souknev, Joukow-Werejnikow, & Kasanzeva, 877 (Pl.)
Federated Malay States, 220, 834 (Mal.), 458, 460 (Misc.), 880 (B.R.)
Felix, A., 32 (Fev.)
Feng, L. C., 125 (Hel.), 486 (Misc.)
—— & Yao, K. F., 125 (Hel.)
Fenjuk, B. & Demjashev, M., 872 (Pl.)
Fermoselle Bacardi, J. J., with Portuondo del Pino, 585 (Hel.)
Fernandez, A. J., 272 (Mal.)
Fernandez, J. M. M., 607 (Lep.)
—— & Schujman, S., 311 (Lep.)
Fernandez Valdes, A., with Gimeno de Sande, 601 (Lep.)
Fernando, P. B. & Sandarasagara, A. P., 687 (Mal.)
—— & Wijerama, E. M., 275 (Mal.)
Fernando, S. E., 140 (Der.)
Feron, (945) (Lep.)
Ferroir, J., with Mollaret, 351 (Lept.)
Fialho, A., 145 (Der.)
Field, J. W. & Niven, J. C., 798 (Mal.)
Findlay, G. M. & Davey, T. H., 615 (Y.F.)
—— & Mahaffy, A. F., 342 (Y.F.)
——, Stefanopoulo, G. J., Davey, T. H. & Mahaffy, A. F., 339 (Y.F.)
—— & Stern, R. O., 340 (Y.F.)
Finlayson, M. H., 402 (Sn.)
Fischl, V., 614 (Lep.)
Fisk, R. T., with Hoyt & Moore, 323 (Rab.)
Flecker, H., 722 (Misc.)
Fleischer, D., 405 (Diet.)
Fletcher, A. H., 256 (Mal.)
Fletcher, W., 32 (Fev.)
Flippin, H. F., 927 (Fev.)
da Fonseca, F., 424 (Fev.), 502 (K.A.)
Forkner, C. E. & Zia, L. S., 16 (K.A.)
——, with —— 16 (K.A.)
Fossen, A., 883 (B.R.)
——, with Müller, 156 (Misc.)
Foster, A. O., 119 *bis* (Hel.),
Fourès, R., with Benhamou, 503 (K.A.)
Fourie, L., 871 (Pl.)
Fourrier, J., Genova, A. & Duverger, M., 500 (K.A.)
Fox, H., 140, 144 (Der.)
Foy, H. & Kondi, A., 511 (Bl.), 549 (Hel.)
——, —— & Peristeris, M., 793 (Mal.)

Franchi, F. & Sautet, J., 270 (Mal.)
Franchini, G., 468 (Misc.)
Franco, E. E., 502 (K.A.)
Fraser, A. G. L., with Cunningham, 54 (R.F.)
Fraser, N. D., 298, 943 (Lep.)
Fraser, W. A. C., (137) (Hel.)
Freeborn, S. B. & Berry, L. J., 637 (Misc.)
de Freitas, O., 114 (Hel.)
French, M. H., 187 (S.S.)
—— & Hornby, H. E., 194 (S.S.)
Fréville, L. H., 309 (Lep.)
—— & Nguyen-van-Luong, (769) (Mal.)
Fribourg-Blanc, with Lhermitte, 621 (Y.F.)
Fróes, H. P., 84 (B.R.)
Früauf, V., with Gnutenko, 917 (Fev.)
Fryer, S. V., 322 (Rab.)
Funayama, J., 317 (Rab.)
Fusco, G. & Chionetti, U., 630 (Misc.)

G.

Gabaldon, A., 712, 714 (Dys.)
Gabriel, P., with Marie, 350 (Lept.)
Gabrieljan, M. J., 90 (Hel.)
Galliard, H. & Sautet, J., 242 (Mal.)
Gambia, 337 (Y.F.)
Ganguli, A. C., 286 (Mal.)
Ganguli, S. K., with Chopra & Sen, 275 (Mal.)
Garcia, E. Y., 142 (Der.), 532 (Am.)
—— & Africa, C. M., 587 (Hel.)
——, with —— 91, 104 (Hel.)
——, with —— & Layco, 131 (Hel.)
——, with —— & de Leon, 90, 560 *bis*, (Hel.)
Garnham, P. C. C., 262 (Mal.)
Gaschen, H., with Genevray, Autret & Dodero, 489 (Misc.)
——, with Morin & Nguyen-Dinh-Hao, 836 (Mal.)
——, with Raynal, 850 (Mal.)
Gater, B. A. R., 497 (B.R.)
Gaud & Nain, 435 (Fev.)
Gaud, M., 18 (K.A.) 34, (926) (Fev.)
Gauducheau, A., 532 (Am.)
Gavrilov, W., & Dubois, A., 610 (Lep.)
——, with Rodhain, 595 (Hel.)
Gear, H. S., 837 (Mal.)
Genevray, J. & Dodero, J., 321 (Rab.)
——, Gaschen, H., Autret, M. & Dodero, J., 489 (Misc.)
Genova, A., with Fourrier & Duverger, 500 (K.A.)
Geracitano, A., 239 (Mal.)
Gerlach, F. & Schweinburg, F., 320, 746 *bis* (Rab.)
Gerlach, J. H. A., 261 (Mal.)
—— with Wolff, 140 (Der.)
Germain, A., with Plazy, 41 (Fev.)
Germinal, with Mazza & Basso, 207 (S.S.)
Germond, R. C., 603, 935 (Lep.)
Gessner, O. & Esser, W., 492 (Misc.)
Ghosh, B. M., with Krishnan & Bose, 521 (Bl.)
Ghosh, B. N., 813 (B.R.)
—— & Sinton, J. A., 239 (Mal.)
Ghosh, H., 865 (Chl.)
Gibson, P. C., with Baker, 392 (Sn.)
Giemsa, G., 80 (Misc.)
Giglioli, G., 801 (Misc.)
Gilbert, A. P. W., with Soesilo, 231 (Mal.)
Gil Collado, J., 346 (Y.F.)
Gill, C. A., 172 (S.S.), 214, 682, 687 (Mal.)
Gille, R., with Benhamou, 505 (K.A.), 782 (Mal.)
Gillespie, J. O., 54 (R.F.)
Gimeno de Sande, A. & Fernandez Valdes, A., 601 (Lep.)
Gingrich, W., 280 (Mal.)
——, with Dawson & Hollar, 272 (Mal.)
Ginsburg, J. M., 248 (Mal.)
Giordano, M., (94) (Hel).
Giovannola, A., 954, (955) *bis* (Hel.)
Girard, G., 366, 370, 868 (Pl.)
—— & Robic, J., 877 (Pl.)
Giraud, Berthier, Ciaudo & Praliaud, 22 (K.A.)
—— & Ciaudo, 23 (K.A.)
——, —— & Bernard, R., 503, 504, 509 (K.A.)
Girolami, M., 553 (Hel.)
Giroud, P., 43, 423, 430, 431, 926 *bis* (Fev.)
—— & Haber, P., 45 (Fev.)
—— & Plotz, H., 42, 43, 438 (Fev.)
——, with ——, 916 *bis* (Fev.)
Githens, T. S., 379 (Sn.)
Giunta, G., 49 (Fev.)
Glaser, R. W. & Coria, N. A., 711 (Dys.)
Glyn-Hughes, F., with Yorke, Murgatroyd, Lester & Ross, 659 (S.S.)
Gnochvili, I., Keigueloukhes, I. & Mourgiri, M., 780 (Mal..
Gnutenko, M. & Früauf, V., 917 (Fev.)
Godding, H. C., 751 (Rab.)
Gögl, H., 565 (Hel.)
Goldfeder, A. E., 775 (Mal.)
Goldie, H., 200 (S.S.)
Goldwater, L. J., Steinberg, I., Möst, H. & Connery, J. E., 122 (Hel.)
Golob, M., 706 (Am.)
Golovine, S., (614) (Lep.)
Gomes, J. M., 303 (Lep.)
Goodman, C., 42 (Fev.)
—— & Brodie, M., 42 (Fev.)
Googe, J. T., with Keller, Cotrell, Miller, Jr. & Harvey, 583 (Hel.)
Goosmann, C., (784) (Mal.)
Gordon, A., with Orenstein, 567 (B.R.)
Gordon, J. E. & Hughes, T. P., 624 (Y.F.)
Gordon, R. M. & Davey, T. H., 514 (Bl.)
Gorrie, R., with Waite, 136 (Hel.)
Gotay, J. B., Costa Mandry, O. & Payne, G. C., 969 (Y. & S.)
Gouget, R., (263) (Mal.)
Gouttas, A., with Bensis, 516 (Bl.)
Govi, L., with Mazza, 208 (S.S.)
Govindaswamy, M. V., 795 (Mal.)
Grado, G., 102 (Hel.)
Graef, I., with Clark, 96 (Hel.)
Graham-Yooll, M. A., 295 (Lep.)
Gramberg, K. P. C. A., (945) (Lep.)
Grasset, E. & Zoutendyk, A., 382 (Sn.)
——, —— & Schaafsma, A., 383 (Sn.)
Grayson, W. B. & Hastings, G., 326 (Rab.)
Greco, Z., with Bogliolo, 22 (K.A.)
Greenwald, H. M., 395 (Sn.)
Greval, S. D. S., 750 (Rab.)
Grevenstuk, A., (725) (Misc.)

Grey, J. C. P., 870 (Pl.)
Grieco, V., 939 (Lep.)
Griffitts, T. H. D. & Michel, C., 344 (Y.F.)
Grillo, J., with Schlossberger, 188 (S.S.)
—— & Schmitz, J., 189 (S.S.)
Grimard, L., with Nattan-Larrier, 19, 21 (K.A.)
Gritti, P., 17 (K.A.)
Grossman, J. & Solomon, E., 776 (Mal.)
Gruhzit, O. M., with W. D. Lindsay, G. Hendricks & M. C. Dodd, 673 (S.S.)
Grunske, F., 809 (B.R.)
Grütz, O., 142 (Der.)
Guerricchio, A., 18 (K.A.)
Guha, P. K., 29 (K.A.)
Guillain, G. & Lereboullet, J., 351 (Lept.)
Guinto, R., with Doull, Rodriguez & Plantilla, 931 (Lep.)
Gunther, C. E. M., 41 (Fev.)
Gupta, B. M. D., 490 (Misc.)
Gupta, C. R. D., with Napier, 75 (Misc.)
Gupta, J. C., with Chopra & Roy, 492 (Misc.)
——, with —— & Sen, 795 (Mal.)
Gupta, P. K. S., (270) (Mal.)
Gupta, S. K., with Pasricha & de Monte, 865 (Chl.)
Gurkirpal Singh, with Taylor & Ahuja, 377 (Chl.)

H

Habachi, S., with Meyerhof, 449 (Oph.)
Haber, P., with Giroud, 45 (Fev.)
Hackett, C. J., 962 *bis* (Y. & S.)
Hackett, L. W. & Lewis, D. J., 848 (Mal.)
Hager, F. D., with Zinsser & Castaneda, 429 (Fev.)
Hai, with Mickaniewski, (769) (Mal.)
Hakansson, E. G., 534 (Am.)
Halder, K. C., with Smith, 26 (K.A.)
Halder, M. N., with Chatterjee, 408 (Diet.)
Hall, M. C., 950 (Hel.)
Hameed, A., 631 (Misc.)
Hamid, E. A., (278) (Mal.)
Hamilton, A. H., 160 (B.R.)
Hamilton, T. D., with Harvey, 479 (Misc.)
Hansmann, G. H. & Schenken, J. R., 143 (Der.)
Hanson, H., 280 (Mal.), 929 *bis* (Fev.)
Happold, F. C. & Stephenson, D., 489 (Misc.)
Harbhagwan, with Sinton, 282 (Mal.)
Harrison, R. W., with Julianelle, 449 *bis* (Oph.)
Harvey, D., 33 (Fev.)
Harvey, R. H., with Keller, Googe, Cottrell, & Miller, Jr., 583 (Hel.)
Harvey, W. F. & Hamilton, T. D., 479 (Misc.)
Haslé, G., Toullec, F. & Vaucel, M., 61, 353 (Lept.)
——, with Vaucel, 474 (Misc.)
Hastings, G., with Grayson, 326 (Rab.)
Hauer, A., 523 (Bl.)
Hay, D. C., Spaar, A. E. & Ludovici, H. L., 791 (Mal.)
Hazen, E. L., with Maillard, 47 (Fev.)
Headlee, T. J., 486 (Misc.)
Headlee, W. H., 121 (Hel.).
——, with Faust, (551) (Hel.)
Health Briefs, (761) (Mal.)
Heathman, L. S., 589 (Hel.)
Hegner, R. & Eskridge, L., 713 *bis* (Dys.)
Heiberg, B., 862 *bis* (Chl.)
Heimburger, L. F., with Cowdry, 301 (Lep.)
——, with —— & Williams, 605 (Lep.)
Hemenway, R. V., with Dang & Lau, (777) (Mal.)
Henderson, W. W., 172 (S.S.)
Hendricks, G., with Gruhzit, Lindsay & Dodd, 673 (S.S.)
Henry, A. F. X., 329 (B. R.)
Henry, E., with Rodhain, 671 (S.S.)
Henry, T. A., 785 (Mal.)
Hermans, E. H., with Breijer & Elsbach, 964 (Y. & S.)
Hermant, 39 (Fev.)
Herms, W. B. & Wheeler, C. M., 347, 348 (R.F.)
——, with —— & Meyer, 55 (R.F.)
Hernando, E., (860) (Chl.)
Herrada Llibre, M., with Arenas Martorell, 324 (Rab.)
Herrada, M., with Arenas & Alvarez, 951 (Hel.)
Herrero Rubio, P., 499 (K.A.)
Herrick, C. A. & Cross, S. X., 669 (S.S.)
Hetsch, H. & Anton, H., (710) (Dys.)
Hewlett, R. F. L., with Tanner, 264 (Mal.)
Hicks, E. P., 235 (Mal.)
—— & Chand, S. D., 616 (Y.F.)
Hidajat, M. D. & Essed, W. F. R., 699 (R.B.F.)
Hijman, A. J. & van Veen, A. G., 724 (Misc.)
Hill, R. B. & Olavarria, J., 270 (Mal.)
Hingst, H. E., (247) (Mal.)
Hinman, E. H., 132, 133, 595 (Hel.)
—— & Baker, D. D., (951) (Hel.)
Hirsch, R., with Canciulesco, (783) (Mal.)
Hoare, C. A., 647 *bis* (S.S.)
Hoeppli, R., 110 (Hel.)
Hoff, F. & Sauerstein, H., 562 (Hel.)
Hoffmann, W. H., (614), 939 (Lep.)
Holiday, E. R., Kerridge, P. M. T. & Smith F. C., 478 (Misc.)
Holland, E. M., 931 (Lep.)
Hollar, E. D., with Dawson & Gingrich, 272 (Mal.)
——, with Sharp, 52 (Fev.)
Homma, H., with Kurauchi, 877 (Pl.)
van Hoof, L., 645 (S.S.)
d'Hooghe, M., 134 (Hel.)
Hooper, R. C. D. & Loewenthal, L. J. A., 716 (Misc.)
Hoops, A. L., 790 (Mal.)
Hopkins, G. H. E., 359 (Pl.), 570 (B.R.)
Horn, L., with Brandt, 268 (Mal.)
Hornby, H. E., 193 (S.S.)
——, with French, 194 (S.S.)
Hoskins, M., 341 *bis* (Y.F.)
Ho-thieu-Ngan, with Ragiot, Delbove & Nguyen-van-Huong, 354 (Lept.), 433 (Fev.)
Houssiau, F. & Du Soleil, G., 156 (Misc.)
Howard, A. C., 599 (Lep.)
Howat, C. H., with Lamborn, 654 (S.S.)
Hoyt, A., Fisk, R. T. & Moore, F. J., 323 (Rab.)
Hoyt, R. N. & Worden, R. D., 262 (Mal.)

Hu, S. M. K., 124 *bis* (Hel.), 254, 255 (Mal.)
——, with Toumanoff, 849 (Mal.)
Huard, P. & May, M., (547) (Am.)
—— & Meyer-May, J., 727 (B.R.)
——, with ——, 536 (Am.)
——, Renucci, N. & Huynh-Tan-Doi, 536 (Am.)
Hudson, E. H., 966 (Y. & S.)
Hueck, O., 308 (Lep.)
Huet, P. C., with Laignel-Lavastine & Koressios, 393 (Sn.)
Huff, C. G., 237, 277 (Mal.)
Hughes, J. C., Diethelm, O. A. & Tebbutt, A. H., 914 (Fev.)
Hughes, T. P., with Gordon, 624 (Y.F.)
Huizenga, L. S., (315) (Lep.)
Hulse, C. R., with O'Connor, 592 (Hel.)
Humphreys, R. M. & Mayne, F. S., 509 (K.A.)
Hunnicutt, T. N., Jr., 98 (Hel.)
Hunninen, A. V., 99, 563 (Hel.)
van Hunsel, J. H. F. E., 859 (Chl.)
Hurst, E. W., 745 (Rab.)
Hurwitz, E. & Anderson, H. H., 934 (Lep.)
Huynh-van-Huy, with Dorolle, Ngo-Quang-Ly & Tran-van-Tam, 612 (Lep.)
Huynh-Tan-Doi, with Huard & Renucci, 536 (Am.)

I

Icaza, J. I. 369 (Pl.)
Ide, K., 559 *bis* (Hel.)
Iglesias, A. S., 950 (Hel.)
Ildrim, D. J., 138 (Der.)
Iljin, B., with Tikhomirova & Sagorskaja, 368 (Pl.)
Inclan, C. & Santamarina, V., 481 (Misc.)
Indian Medical Gazette, 107 (Hel.), 406 (Diet.) 792 (Mal.)
Ingersoll, W., 800 (Misc.)
Inhelder, H. E., (110) (Hel.)
Innes, J. R., (934) (Lep.)
Inoue, K., with Sumi, 533 (Am.)
International Journal of Leprosy, Manila, 600 (Lep.)
Ioff, I., with Sassuchin & Tiflow, (879) (Pl.)
Iraq, 762 (Mal.)
Isabolinski, M., Lewzow, I. & Tschernjak, I., 317 (Rab.)
Istamanjan, L. S., with Sadojan, 259 (Mal.)
Ivanić, M., 778 *bis* (Mal.)
Iwanowsky, N. & Faddeeva, T., 876 (Pl.)
Iwata, S., 98 (Hel.)
Iyengar, K. R. K., 321 (Rab.)
Iyengar, M. O. T., 594 (Hel.)
Izaguirre, A., with Cáceres, 209 (S.S.)
Izumi, M., 561 (Hel.)

J

Jackson, R., 239 (Mal.)
Jackson, R. B., 260 (Mal.)
Jackson, S., 514 (Bl.)
Jacobsthal, E., 779 (Mal.)
Jacocks, W. P., Kendrick, J. F. & Sweet, W. C., 112 (Hel.)
Jacono, I., with Castellani, 139 (Der.)
Jadin, J., with Bruynoghe, 429 (Fev.)
Jamaica, 625 (Misc.)
James, J. F., 488 (Misc.)
James, S. P., 213, 228, 857 (Mal.), 333 (Y.F.)
——, Nicol, W. D. & Shute, P. G., 684 (Mal.)
v. Jancsó, N. & v. Jancsó, H., 204 (S.S.)
—— & Novák, E., 56 (R.F.)
Janzi, E. Z., with Mazza & Benitez, 676 (S.S.)
Jensen, M. H., with Keller & Leathers, 581 (Hel.)
Jeremy, R. & Jones, E. B., 90 (Hel.)
de Jesus, Z., (559) (Hel.)
Jettmar, H. M., 79 (Misc.)
Johnson, C. M. & De Rivas, G. T., 675 (S.S.)
Johnson, V. M., with Sayad & Faust, 596 (Hel.)
Johnstone, H. G., with Reed, 63 (Sp.)
Joltrain, E., (874) (Pl.)
Jones, E. B., with Jeremy, 90 (Hel.)
Jones, T. L. & Kingscote, A. A., 106 (Hel.)
Jonnesco, D., 747 (Rab.)
——, with Proca, 744 (Rab.)
Jordan, P., 611 (Lep.)
Jorge, A. L., da Silva, J. F. & Leal, A. E., 266 (Mal.)
Jorge, R., 18 (K.A.), 357 (Pl.)
Jouck, N., 711 (Dys.)
Joukow-Werejnikow, N. & Faworissowa, B., 371 (Pl.)
——, with Souknev, Faworissowa & Kasanzava, 877 (Pl.)
Journal of the Royal Army Medical Corps, 476 (Misc.)
Joyeux, C. & Baer, J. G., 86 (Hel.)
——, Sédan, J. & Esménard, J., 135 (Hel.)
——, with Zwirn & Aboucaya, 109 (Hel.)
Julianelle, L. A. & Harrison, R. W., 449 *bis* (Oph.)
Junior, J. de M., 314 (Lep.)
Junior, R., 937 (Lep.)
Juster, I. R., 479 (Misc.)

K

Kabler, P., 527 (Am.)
Kalabuchov, N. & Raevsky, W., 872 (Pl.)
Kalantarian, H. W., 548 (Hel.)
Kan, H. & Kung, J., (955) (Hel.)
Kan, H. C., 956 (Hel.)
Kanarewskaja, A. A. & Bogomolow, I. L., 434 (Fev.)
Kanazawa, K., Yaoi & Sato, 743 (Rab.)
Kanda, K., 448 (Oph.)
Kaneko, K., 60 (Lept.)
K'ang, H. J., with Ch'eng, 564 (Hel.)
Kapur, N. C., 378 (Chl.)
Kapusto, M. L., 200 (S.S.)
Karamchandani, P. V., 124 (Hel.)
Karapetjan, E. G., (275) (Mal.)
Kariadi, (797) (Mal.)
Karunaratne, W. A. E., 720 (Misc.)
——, with Cameron, 586 (Hel.)
Kasanzeva, E., with Souknev, Joukow-Werejnikow & Faworissowa, 877 (Pl.)
Kassirsky, I. A., 31 (K.A.)
Kattan, H., 763 (Mal.)
Kau, Z. M., 594 (Hel.)
Kawamura, K., 927 (Fev.)
Kawana, H., with Komiya & Tao, 558, 948 *quat.* (Hel.)

Kedrowsky, W. I., 598 (Lep.)
Kehar, N. D., 796 *bis* (Mal.)
Keigueloukhes, I., with Gnochvili & Mourgiri, 780 (Mal.)
Keil, E., (301) (Lep.)
Kellaway, C. H. & Williams, F. E., 385 (Sn.)
Keller, A. E., Googe, J. T., Cottrell, H. B., Miller, D. G., Jr. & Harvey, R. H., 583 (Hel.)
—— & Leathers, W. S., 573 (Hel.)
——, with ——, 580 (Hel.)
——, —— & Jensen, M. H., 581 (Hel.)
Kellersberger, E. R., 661 (S.S.)
Kellogg, W. H., 363 (Pl.)
Kemp, H. A., Moursund, W. H. & Wright, H. E., 347 (R.F.)
Kenawy, M. R., with Day, 951 (Hel.)
Kendrick, J. F., with Jacocks & Sweet, 112 (Hel.)
Kennedy, W. P. & Mackay, I., 480 (Misc.)
Kenya Colony & Protectorate, 465 (Misc.)
Kerr, K. B., 104 (Hel.)
Kerridge, P. M. T., with Holiday & Smith, 478 (Misc.)
Kevorkov, N. P., with Chodukin & Petrov, 500 (K.A.)
——, with —— & Sofiev, 506 (K.A.)
Keys, S., with Thomas, 776 (Mal.)
Khalil, M., 17 (K.A.), 127, 957 *bis* (Hel.), 450 (Oph.)
—— & Azim, M. A., 92, 953 (Hel.)
—— & Salah, M., 112 (Hel.)
Khaw, O. K., 97 (Hel.)
Kikuth, W. & Verfürth, D. H., 301 (Lep.)
King, P. Z., with Liu, 454 (Misc.)
King, W. V. & Del Rosario, F., 853 (Mal.)
Kingsbury, A. N., 220, 834 (Mal.), 460 (Misc.), 880 (B.R.)
Kingscote, A. A., with Jones, 106 (Hel.)
Kirby-Smith, J. L., 117 (Hel.)
Kirk, J. B., 462 (Misc.), 554 (Hel.)
—— & Cantin, A. Y., 108 (Hel.)
Kirwan, E. O'G., 451, 904 (Oph.)
Kitabatake, E. & Liu, K. P., 473 (Misc.)
Kitchen, S. F., with Boyd, 842 (Mal.)
——, with —— & Mulrennan, 843 (Mal.)
——, with —— & Stratman-Thomas, 240, 842 *bis* (Mal.)
Kligler, I. J., Aschner, M. & Levine, S., 427 (Fev.)
—— & Berman, M., 667 (S.S.)
—— & Comaroff, R., 205 (S.S.)
von Klobusitzky, D., 380, 381 (Sn.)
Knipfer, A., 147 (Der.)
Knott, J., 129 (Hel.), 602 (Lep.)
Knowles, R., 840 (Mal.)
——, Das Gupta, B. M. & Sen, S., 700 (R.B.F.)
Ko, B., with To, 561 (Hel.)
Kobashi, S., with Nakamura & Matsumoto, 306 (Lep.)
Kodama, M., 42 (Fev.)
Kodyat, R., with Sitanala, 934 (Lep.)
Koechlin, D., with Marchoux & Chorine, 57 (R.F.), 307 (Lep.)
Kofoid, C. A., McNeil, E. & Bonestell, A. E., 80 (Misc.)
——, Wood, F. D. & McNeil, E., 677 (S.S.)
Kohls, G. M. & Cooley, R. A., 693 (R.F.)
Koiter, D., 701 (Am.)
Koman Nayar, K., with Wright, 450 (Oph.)
Komiya, Y., Kawana, H. & Tao, S. C., 558, 948 *quat.* (Hel.)
Komp, W. H. W. & Clark, H. C., 835 (Mal.)
Kondi, A., with Foy, 511 (Bl.), 549 (Hel.)
——, with —— & Peristeris, 793 (Mal.)
Kopciowska, L., 317 (Rab.)
Koressios, N. T., with Laignel-Lavastine & Huët, 393 (Sn.)
Korobkova, E., 863 (Chl.)
Korovitzky, L. K., with Tscherkes & Litvack, 805 (Misc.)
Kotorii, S., 59, 60 (Lept.)
Kouri, P., 88 (Hel.)
—— & Basnuevo, J. G., 632 (Misc.)
——, —— & Arenas, R., 587 (Hel.)
——, ——, Alvaré, L. & Lescano, O., 88, 558 (Hel.)
——, with Bolaños, Anido & Basnuevo, 558 (Hel.)
——, with Calvo Fonseca, R. & Basnuevo, J. G., 550 (Hel.)
——, with —— & ——, 108, 550 (Hel.)
—— & Del Frade, A., 87 (Hel.)
—— & Sellek, A., 588 (Hel.)
—— & Valverde, A., 89 *bis* (Hel.)
Kourilsky, R. & Mamou, H., 352 (Lept.)
Kousa, 509 (K.A.)
Kouwenaar, W. & Wolff, J. W., 442 (Fev.)
——, with ——, 49, 421 (Fev.)
Kraan, H., de Buck, A. & Swellengrebel, N. H., 288 (Mal.)
Krauel, G., with Budelmann, 518 (Bl.)
Krause, M., with Collier, 192 (S.S.)
Krishnan, K. V., Chopra, R. N. & Mukherjee, S. N., 79 (Misc.)
——, Ghosh, B. M. & Bose, P. N., 521 (Bl.)
—— & Pai, N. G., 517 (Bl.)
Krishnaswami, T. K., with Menon, 81 (Misc.)
—— with —— & Annamalai, 505 (K.A.)
Kriz, J. R., 939 (Lep.)
Kröber, F., 694 (R.F.), 786 (Mal.)
Kubo, M., 517 *bis* (Bl.), 704 (Am.), 788 (Mal.)
Kubota, K., (373) (Chl.)
Kumm, H. W., 961 (961) (Y. & S.)
——, with Saunders & Rerrie, 960 (Y. & S.)
—— & Turner, T. B., 961 (Y. & S.)
Kunert, H., with Schilling, Schreck & Neumann, 195, 664 (S.S.)
Kung, J. C., with Kan, (955) (Hel.)
Kuo, K. W., Takahara, K., Adachi, J. & Saito, K., 627 (Misc.)
Kurauchi, K. & Homma, H., 877 (Pl.)
Kuroda, O., 928 (Fev.)
Kuypers, C. A., 432 (Fev.)
Kuznetsov, N. J., 368 (Pl.)
Kyu, K., with To, 546 Am.)

L

Laake, E. W., with Parish, 488 (Misc.)
Labbé, M., Boulin, Uhry & Ullman, 351 (Lept.)
La Cava, G., 263 (Mal.)
Lafuma, J., with Meersseman, (784) *bis* (Mal.)
Lagoudaky, S., 609 (Lep.)

Lahiri, S. C., 373 (Chl.)
Lai, D. G. & Chen, W. Y., 941 (Lep.)
——, Li Y.-J. & Chang, W., 254 (Mal.)
Laignel-Lavastine, Huët, P. C. & Koressios, N. T., 393 (Sn.)
Laigret, 620 (Y.F.)
Laigret, J., 339 (Y.F.)
—— & Boneau, E., 339 (Y.F.)
—— & Durand, R., 427 (Fev.)
——, with Nicolle, 34, 437, 619, (Fev.)
——, with Sellards, 618 (Y.F.)
Lal, S., with Pasricha & Bannerjee, 924 (Fev.)
Lambert, S. M., 583 (Hel.), (963) (Y. & S.)
Lamborn, W. A. & Howat, C. H., 654 (S.S.)
Lambusta, A., 835 (Mal.)
Lampe, P. H. J. & de Moor, C. E., 307, 945 (Lep.)
Lamprell, B. A., with Ramsay & Chandra, 843 (Mal.)
Lamson, P. D. & Brown, H. W., 577 (Hel.)
Lancet, 555 (Hel.), 638 (Misc.)
Lande, P. & Sigalas, R., 577 (Hel.)
Lane, C., 129 (Hel.)
Langan, A. M., & Mathew, R. Y., 41 (Fev.)
Langdon-Brown, W., 72 (Misc.)
de Langen, C. D., 71, 156 (Misc.)
—— & Djohan, B., 409 (Diet.)
——, with Lichtenstein, 161 (B.R.)
—— & Storm, C. J., (234) (Mal.)
Lannelongue, G., with Sabrazès, Dutrénit & Bonnel, 353 (Lept.)
Lara & de Vera, (604) (Lep.)
Larrousse, F., 566 (Hel.)
Larsen, A. E., 454 (Misc.)
Larthe, N., with Sazerac, 664 (S.S.)
Lasnet, 39 (Fev.)
Latychev, N., 349 (R.F.)
Lau, S., with Dang & Hemenway, (777) (Mal.)
Launoy, L., 203 (S.S.)
——, Prieur, M. & Ancelot, A., 203 (S.S.)
Lavergne, J., 114 (Hel.)
——, with Daléas, 783 (Mal.)
Laverick, J. V., 699 (R.B.F.)
Lavier, G., with Brumpt, 80 (Misc.)
Lawrence, H., 145, 147 (Der.)
Lawson, H. A., with Castle, Rhoads & Payne, 66 (Sp.)
Layco, J., with Africa & Garcia, 131 (Hel.)
Leal, A. E., with Jorge & da Silva, 266 (Mal.)
Leathers, W. S. & Keller, A. E., 580 (Hel.)
——, with ——, 573 (Hel.)
——, with —— & Jensen, 581 (Hel.)
Le Bihan, A., 746 (Rab.)
Le Chuiton, F., Berge, C. & Pennanéachh, J. 439, 927 (Fev.)
——, Mondon, H., Berge, C., Pennanéac'h, J. & Dubreuil, J., 438 (Fev.)
Ledentu, G., 469 (Misc.)
Lee, C. U. & Chu, C. F., 30 (K.A.).
—— & Chu, H. J., 96 (Hel.)
—— & Chung, H. L., 97 (Hel.), 503 (K.A.)
—— with ——, 103 (Hel.)
Lee, D. H. K., 628 (Misc.)
Leeson, H. S., 879 (Pl.)
Lefrou, G., 606, 942 (Lep.)
—— & Des Essarts, J. Q., 606, 936 (Lep.)
——, with ——, 298 (Lep.)
Lega, G., with Raffaele, 844 (Mal.)
Le Gac, P., Espian, M. & Rarivoson, A., 952 (Hel.)
Le Gall, Seyberlich & Brault, 874 (Pl.)
Legroux, R. & Lwoff, A., 238 (Mal.)
Leigh, O. C., with Drinker & Augustine, 129 (Hel.)
Leiper, R. T., 947 (Hel.)
Lenke, S. E., with Rosenfeld, 393 (Sn.)
Lentze, F. A., 121 (Hel.)
Leo, T. L., 92 (Hel.)
de Leon, W., with Africa & Garcia, 90, 560 *bis* (Hel.)
Lépine, P., 316, 321 (Rab.), 432, 436 (Fev.)
—— & Bilfinger, F., 508 (K.A.)
—— & Cruveilhier, L., 317 (Rab.)
—— & Lorando, N., 46, 912 (Fev.)
—— & Markianos, J., 612 (Lep.)
—— & Sautter, V., 322 (Rab.), 430 (Fev.)
Le Port, L. R., 177 (S.S.)
Leprosy in India, 293, 599, 933 (Lep.)
Leprosy Review, 292, 293, 598, 932 (Lep.)
Lereboullet, J., with Guillain, 351 (Lept.)
Lescano, O., with Kouri, Basnuevo & Alvare, 88, 558 (Hel.)
Lester, H. M. O., 169 (S.S.)
——, with Yorke, Murgatroyd, Glyn-Hughes & Ross, 659 (S.S.)
Lestoquard, F. & Donatien, 507 (K.A.)
——, with ——, 20 *bis* (K.A.), 46, 925 (Fev.)
Levaditi, C. & Deutsch, V., 57 (R.F.)
—— & Schoen, R., 318, 319 *bis*, 750 (Rab.)
Le-van-Phung, with Montel, 612 (Lep.)
——, with ——, Nguyen-van-Khai, Tran-van-Hanh, Do-van-Hoanh, Truong-van-Que & Nguyen-ngoc-Nhuan, 309 (Lep.)
Levine, S., with Kligler & Aschner, 427 (Fev.)
Lévy, H., with Sergent, 348 (R.F.)
Lewis, D. J., with Hackett, 848 (Mal.)
Lewis, S. J., 423 (Fev.)
Lewthwaite, R., 433 (Fev.)
—— & Savoor, S. R., 424, 911, 921, 922 (Fev.)
Lewzow, I., with Isabolinski & Tschernjak, 317 (Rab.)
Lhérisson, C., 467 (Misc.)
Lhermitte, J. & Fribourg-Blanc, 621 (Y.F.)
Li, C. C., with Chang & Young, 228 (Mal.)
Li, Y. J., with Lai & Chang, 254 (Mal.)
Lichtenstein, A., with de Langen, 161 (B.R.)
Liddo, S. & Bogliolo, L., 28 (K.A.)
Lie, H. P., 603, 605 (Lep.)
Lillie, R. D., 441 (Fev.)
Lima, C. de F., 316 (Rab.)
Lima, E. de Q., with Torres, 743 (Rab.)
Lima, L. de S., 304, (315) (Lep.)
Lindberg, K., (136), 596 *bis* (Hel.)
Lindeman, S. J. L. & Lyburn, R. St. J., 103 (Hel.)
Lindsay, W. D., with Gruhzit, Hendricks & Dodd, 673 (S.S.)
Ling, L. C., 247 (Mal.)
Link, T., 391 (Sn.)
Linton, R. W., Mitra, B. N. & Mullick, D. N., 375 (Chl.)
——, —— & Seal, S. C., (376) (Chl.)
Lintwaréff, J. J., 778 (Mal.)
Lipscomb, F. M., 565 (Hel.)
Lipstein, I., 694 (R.F.)
Lister, S., 82 (B.R.)

Litvack, J. I., with Tscherkes, L. A. & Korovitzky, 805 (Misc.)
Liu. J. H. & King, P. Z., 454 (Misc.)
Liu, K. P., with Kitabatake, 473 (Misc.)
Livierato, S. G. & Salata, C. C., (784) (Mal.)
Livon, J., with Violle, 744 (Rab.)
Li Yuan Po, 693 (R.F.)
Lloyd, B. J., 616 (Y.F.)
Lloyd, H. M., 653 (S.S.)
Lloyd, W., 333 (Y.F.)
—— & Mahaffy, A. F., 624 (Y.F.)
——, Theiler, M. & Ricci, N. I., 623 (Y.F.)
Lobel, L. W. M., 946 (Lep.)
Loeper, M. & collaborators, 327 (B.R.)
Loewenthal, L. J. A., 404 (Diet.)
——, with Hooper, 716 (Misc.)
van Loghem, J. J., 435 (Fev.)
Loh, V. T. & Tai, T. Y., 860 (Chl.)
Lombardo, V., 538 (Am.)
Long, J. D., 878 (Pl.)
Longmans' African Hygiene Pictures, 167 (B.R.)
Lorando, N., 37 (Fev.)
——, with Lépine, 46, 912 (Fev.)
Loumaigne, with Sanner, 52 (Fev.)
Lourens, L. F. D. E., 320 (Rab.)
Lovett-Campbell, A. C., 555 (Hel.)
Low, G. C., & Manson-Bahr, P. H., with Walters, 585 (Hel.)
Loyola Pereira, O., with de Mello, 308 (Lep.)
Lück, O., 746 (Rab.)
Ludovici, H. L., with Hay & Spaar, 791 (Mal.)
di Lullo, O., 146 (Der.)
Lumbroso, with Rouslacroix & Ciaudo, 506 (K.A.)
Lutrario, A., 18 (K.A.), 223 (Mal.)
Lwoff, A., with Legroux, 238 (Mal.)
Lwoff, M., 79 (Misc.)
Lyburn, R. St. J., with Lindeman, 103 (Hel.)

M

Mabalay, E., with Plantilla, 402 (Sn.), 966 (Y. & S.)
—— with Rodriguez & Tolentino, (601) (Lep.)
MacArthur, W. P., 565 (Hel.)
MacCallan, A. F., 448, 902 (Oph.)
McClamroch, J. M. & Vallotton, J. R., 51 (Fev.)
MacClure, E., 397 (Sn.)
McCoy, O. R., 85, 592 (Hel.)
McEwen, O. R., 72 (Misc.)
Macfarlane, R. G. & Barnett, B., 396 (Sn.)
McGeorge, M., 477 (Misc.)
McGregor, L. J., 763 (Mal.)
McGuire, C., 154 (Misc.)
Macht, D. I., 396 (Sn.)
Macias y Macias, F., 476 (Misc.)
Maciel, H., 568 (B.R.)
Mackay, I., with Kennedy, 480 (Misc.)
Mackay, R., 225 (799) (Mal.)
McKendrick, A. G., 747 (Rab.)
Mackenzie, J. N., 932 (Lep.)
Mackie, F. P., with Fairley, 471 (Misc.)
Mackie, T. T., Miller, D. K., & Rhoads, C. P., 64 (Sp.)
—— & Pound, R. E., 63 (Sp.)
McKinley, E. B., 250 (B.R.)
Mackinnon, J. E., 138 (Der.), 472 (Misc.)
MacLean, 668 (S.S.)
Maclean, G., 173 (S.S.)
McMullin, J. J. A., (541) (Am.)
McNeel, T. E., with Bradley, 486 (Misc.)
McNeil, E., with Kofoid & Bonestell, 80 (Misc.)
——, with —— & Wood, 677 (S.S.)
McRae, A., 106 (Hel.)
Macrez, C., with Troisier & Bariéty, 352 (Lept.)
Madison, R. R., 875 (Pl.)
de Magalhães, O., with Moreira, 40, 915 (Fev.)
Magoon, E. H., 286 (Mal.)
Mahaffy, A. F., with Findlay, 342 (Y.F.)
——, with ——, Stefanopoulo & Davey, 339 (Y.F.)
——, with Lloyd, 624 (Y.F.)
Maillard, E. R. & Hazen, E. L., 47 (Fev.)
Mainoli, M. R., with Mazza, (678) (S.S.)
Mainzer, F., 96 (Hel.)
Majumder, A. R., 540 (Am.)
Malamos, B., 81 (Misc.)
——, with Nauck, 840 (Mal.)
Maldonado, J. G., 941 (Lep.)
Maldonado Sampedro, M., 118 (Hel.)
Mallick, S. M. K., 399 (Sn.)
——, with Shortt, 387 (Sn.)
——, with Taylor, 387 (Sn.)
Mami, A., with Broc, 553 (Hel.)
Mamou, H., with Kourilsky, 352 (Lept.)
Manalang, C., (607), 607 *bis* (Lep.)
Mandekos, A., with Carr & Barber, 759 (Mal.)
Manouélian, Y., 320 (Rab.)
Mansell, R. A., (854) (Mal.)
Manson, D., 625 (Misc.), 794, 840 (Mal.)
Manson-Bahr, P., 160 (B.R.), 709 (Dys.)
—— & Low, with Walters, 585 (Hel.)
Manuwa, S. L. A., 475 *bis* (Misc.)
Manwell, R. D., 237 (Mal.)
Maplestone, P. A., 111 (Hel.)
—— & Mukerji, A. K., 117 (Hel.)
—— & Mukerji, P. K., 575 (Hel.)
—— & Riddle, J. S., 566 (Hel.)
Marañon, J., Perez, A. & Russell, P. F., 232 (Mal.)
Marchal, G., Soulie, P. & Roy, A., 352 (Lept.)
Marchesi, F., 61 (Lept.)
——, Crainz, F. & Scapaticci, R., 18, 508 (K.A.)
—— & Scapaticci, R., 22 (K.A.)
Marchoux, E., Chorine, V. & Koechlin, D., 57 (R.F.), 307 (Lep.)
Marcus, J. M. & Wainer, K. G., 902 (Oph.)
Mariani, G., with Besta, 305 (Lep.)
Marie, J. & Gabriel, P., 350 (Lept.)
Marini, C., with Decourt, Villain & Dupoux, 797 (Mal.)
Marino, S., (536) (Am.)
Markianos, J., with Lépine, 612 (Lep.)
Marmo, A., (714) (Dys.)
Marmorston, J., with Perla, 163 (B.R.)
Marneffe, H., (799) (Mal.)
Marque, E. & Raynal, J., 473 (Misc.)
Marschall, F., 520 (Bl.)
Marseille, A., 265 (Mal.)
Martial, J. E., 271 (Mal.)
Martin, D. S., 473 (Misc.)

Martin, H., 494 (Misc.)
Martin, L. A., with Blanc, 429 (Fev.)
Martin, P. H., 458 (Misc.)
Martin, R., Chorine, V. & Rouessé, G., 30 (K.A.)
——, Rouessé, G. & Bonnefoi, A., 620 (Y.F.)
Martin, R. R., with Rosen & David, 545 (Am.)
Martin del Campo, R., 379 (Sn.)
Martinez, W. H., with Faust, 137 (Hel.)
Martini, E., 253, 846 (Mal.)
Martin Sanchez, A., 697 (R.F.)
Martz, F., 855 (Mal.)
Masayama, 918 (Fev.)
Masoch, T. J., with Beretervide, 535 (Am.)
Mason, M., 164, 882 (B.R.)
Massal, E., 600 (Lep.)
Massias, C., 354 *bis* (Lept.), (433), 913 (Fev.), (547) (Am.)
——, Bourgin, P. & Nguyen-van-Tan, 271 (Mal.)
Massoud, F., 903 (Oph.)
Masunaga, E., with Wayson, 307 (Lep.)
Matewossian, S. M., 530 (Am.)
Mathew, R. Y., 915 (Fev.)
—— with Langan, 41 (Fev.)
Mathis, C., 844 (Mal.)
——, Durieux, C. & Advier, M., 340 (Y.F.)
Mathis, M., 240 (Mal.)
——, with Nicolau & Baffet, 622 (Y.F.)
——, with Rimbaut, 248 (Mal.)
Matsumoto, I., with Nakamura & Kobashi, 306 (Lep.)
Matsunobu, M., 801 (Misc.)
Matsuura, T. (103) (Hel.)
Mattlet, G., 316 (Rab.), 767 (Mal.)
Maurano, F., 313 (Lep.)
Maurice, H., with Sokhey, 367, 876 (Pl.)
Mauritius, 462 *bis*, 717 (Misc.), 554 (Hel.), 763 (Mal.)
Maxcy, K. F., 928 (Fev.)
Maxwell, J. L., 933 (Lep.)
May, J. M., (576) (Hel.)
May, M., with Huard, (547) (Am.)
May, R., 855 (Mal.)
May, R. M., 855 (Mal.)
Mayer, M., with Nocht, 883 (B.R.)
Mayne, B., 280 (Mal.)
Mayne, F. S., with Humphreys, 509 (K.A.)
Mayne, L. C., with Austin, 527, 528 (Am.)
Mazza, S., 676 (S.S.)
——, Benitez, C. & Janzi, E. Z., (676) (S.S.)
—— & Corsi, E. M., (678) (S.S.)
—— & Driollet, E., 211 (S.S.)
——, Germinal & Basso, R., 207 (S.S.)
—— & Govi, L., 208 (S.S.)
—— & Mainoli, M. R., (678) (S.S.)
—— & Nastri de Fischer, C., 209 (S.S.)
—— & Palamedi, B., (678) (S.S.)
—— & Valle, F., (678) (S.S.)
Mededeelingen van den Dienst der Volksgezondheid in Nederlandsch-Indië, 363 (Pl.), 934 (Lep.)
Medulla, C., 96 (Hel.), 348, 349 (R.F.)
Meersseman, F. & Lafuma, J., (784) *bis* (Mal.)
Megaw, J., 32 (Fev.), 858 (Mal.)
Mehta, D. R., with Covell, 909 (Fev.)
Meighan, S. S. & Urquhart, M., 450 (Oph.)
de Meillon, B., 245, 845 (Mal.)
—— & Osburn, H. S., 487 (Misc.)
Meleney, H. E., 280 (Mal.)
—— with Alexander, 528 (Am.)
Mellanby, K., with Duke, 652 (S.S.)
de Mello, A. L. N., 746 (Rab.)
de Mello, F. & Loyola Pereira, O., 308 (Lep.)
de Mello, I. F. & Brás de Sá, L. J., 259 (Mal.)
Meneu, A., with Darder, 431 (Fev.)
Menon, T. B., 127 (Hel.)
—— & Annamalai, D. R., 153 (Misc.)
——, —— & Krishnaswami, T. K., 505 (K.A.)
—— & Krishnaswami, T. K., 81 (Misc.)
Mercier, H., Fabre & Soulage, 910 (Fev.)
——, with Sicé & Robin, 656 (S.S.)
Metivier, H. V. M., 743 (Rab.)
Mettam, R. W. M., 175 (S.S.)
Meyer, J., with Sartory, A., Sartory, R. & Weiss, 144 (Der.)
Meyer, K. F., 433 (Fev.)
—— & Eddie, B., 871 (Pl.)
——, with Wheeler & Herms, 55 (R.F.)
Meyerhof & Habachi, S., 449 (Oph.)
Meyer May, J., 576 (Hel.)
—— & Huard, P., 536 (Am.)
——, with ——, 727 (B.R.)
—— & Vaucel, M., 697 (R.F.)
Michel, C., with Griffitts, 344 (Y.F.)
Mickaniewski & Hai, (769) (Mal.)
Miguel, A. S., with Molina, (19) (K.A.)
Miguelote-Vianna, M., with Vellard, 390 (Sn.)
Miller, D. G., Jr., with Keller, Googe, Cottrell & Harvey, 583 (Hel.)
Miller, D. K., with Mackie & Rhoads, 64 (Sp.)
Millischer, (547) (Am.)
Milne, J. C., (229) (Mal.)
Milne, P. S., with Williams, 637 (Misc.)
Milochevitch, S., with Bordjoški, 139 (Der.)
Mindline, J. & Rosenheim, M. L., 69 (Sp.)
Minning, W., 210 (S.S.)
Missiroli, A., 289, 852 (Mal.)
Mitra, B. N., (376) (Chl.)
——, with Linton & Mullick, 375 (Chl.)
——, with —— & Seal, (376) (Chl.)
Miyake, S., with Takino, 608 (Lep.)
Mochkovski, C., 269, 278, (841) (Mal.)
Mochtar, A. & Sardjito, M., 611 (Lep.)
Moiser, B., 297 (Lep.)
Molina, C. & Miguel, A. S., (19) (K.A.)
Mollaret, P., 569 (B.R.), 624 (Y.F.)
——, with Darré, 619 (Y.F.)
—— & Erber, B., 350 *bis* (Lept.)
—— & Ferroir, J., 351 (Lept.)
—— with Findlay, G. M., 619 (Y.F.)
Mondon, H., with Le Chuiton, Berge, Pennanéac'h, J. & Dubreuil, 438 (Fev.)
Monier, H. & Treillard, M., 291 (Mal.)
Montagne, M. & Rivoalen, A., 369 (Pl.)
Montañes, P. & Carderera, M., 258 (Mal.)
de Monte, A. J. H., with Pasricha & Gupta, 865 (Chl.)
——, with —— & O'Flynn, 866 (Chl.)
Monteiro, J. L., 428 (Fev.)
Montel, M. L. R., (312) (Lep.)
——, Le Van-Phung, Nguyen-van-Khai, Tran-van-Hanh, Do-van-Hoanh, Truong-van-Que & Nguyen-ngoc-Nhuan, 309 (Lep.)
—— & Truong-van-Que, 132 (Hel.), 309 *bis* (Lep.)

Montel, R., 309 *bis*, 944 (Lep.), 914 (Fev.)
——, Bablet, J., Nguyen Ngoc Nhuan & Do van Hoanh, 944 (Lep.)
—— & Le-van-Phung, 612 (Lep.)
—— & Tran-van-Hanh, 310 (Lep.)
—— & Truong-van-Que, 700 (R.B.F.)
Montestruc, E., 836 (Mal.)
—— & Bertrand, C., 127, 557 (Hel.)
Mookerjee, S. L., with Wilson, 493 (Misc.)
de Moor, C. E., with Lampe, 307, 945 (Lep.)
Moore, F. J., with Hoyt & Fisk, 323 (Rab.)
Mora, A. D. & Soares, J. C., 933 (Lep.)
Morales Otero, P., (875) (Pl.)
Morales Villazón, N., with Uriarte, 362, 370 *bis* (Pl.)
——, with —— & Anchezar, 362, 370 (Pl.)
de Moraes, C. V., 272 (Mal.)
Morcos, Z., 531 (Am.)
Moreau, P., with Ragiot, 839 (Mal.)
——, with Sicé, 179 (S.S.)
Moreira, J. A. & de Magalhães, O., 40, 915 (Fev.)
Morimoto, T., 60 (Lept.)
Morin, H. G. S., 761 (Mal.)
——, Gaschen, H. & Nguyen-Dinh-Hao, 836 (Mal.)
Morrison, W. K., 566 (Hel.)
Mosna, E. & Salustri, E., 266 (Mal.)
Most, H., with Goldwater, Steinberg & Connery, 122 (Hel.)
Mostert, H. v. R., 524 (Bl.)
Mosto, D., 539 (Am.)
Mo Ten Sei, 506 (K.A.)
Motta, O. C., 705 (Am.)
Moulinard, M., 958 (Hel.)
Mourgiri, M., with Gnochvili & Keigueloukhes, 780 (Mal.)
Moursund, W. H., with Kemp & Wright, 347 (R.F.)
Moustardier, G., 656 (S.S.)
Muench, H., with Boyd & Stratman-Thomas, 779 (Mal.)
Mühlens, P., (596) (Hel.)
Muir, E., 603, 608 (Lep.)
—— & Chatterji, S. N., 299, 938 (Lep.)
Mukherjee, S. N., with Chopra, 841 (Mal.)
——, with Krishnan & Chopra, 79 (Misc.)
Mukerji, A. K., with Maplestone, 117 (Hel.)
Mukerji, P. K., with Maplestone, 575 (Hel.)
Mulder, J. G. A., 74 (Misc.)
Müller, H. & Fossen, A., 156 (Misc.)
Müller, K. L., 356 (R.B.F.)
Muller, P., 66 (Sp.)
Mullick, D. N., with Linton & Mitra, 375 (Chl.)
Mulrennan, J. A., with Boyd & Kitchen, 843 (Mal.)
Muniz, J. & Regendanz, P., 428 (Fev.)
——, with ——, 427 (Fev.)
Murashima, T., 707 (Dys.)
Murgatroyd, F., with Yorke, Glyn-Hughes, Lester & Ross, 659 (S.S.)
de Muro, P., 273 (Mal.)
Murray, A. J., (53) (Fev.)
Murray, I., 632 (Misc.)
Murthi, K. N., with Pillai, 65 (Sp.)
Musgrave, J. A., 548 (Hel.)
Mustapha, A., 861 (Chl.)

N

Naab, J. P., 30 (K.A.)
Nagahama, M., with Nishiyama, 965 (Y. & S.)
Nain, with Gaud, 435 (Fev.)
Nair, P. K., 107 (Hal.)
Najera Angulo, L., 19, 499 (K.A.)
Nakamura, K., Kobashi, S. & Matsumoto, I., 306 (Lep.)
Nanavati, B. P., with Duggan, 903 (Oph.)
Napier, L. E., 16 (K.A.)
—— & Gupta, C. R. D., 75, 803 (Misc.)
Nastri de Fischer, C., with Mazza, 209 (S.S.)
National Economic Council, 454 (Misc.)
Nattan-Larrier, L. & Dufour, J,, 506, (K.A.)
—— & Grimard, L., 19, 21 (K.A.)
—— & Nouguès, S., 28 (K.A.)
Nauck, E. G. & Malamos, B., 840 (Mal.)
Naumann, H. E., 523 (Bl.)
Nauss, R. W. & Salinger, M. H., 526 (Am.)
Navoka, J. R., 708 (Dys.)
de Negri, U., with Sepulcri, 844 (Mal.)
Negro Vazquez, E., 499 (K.A.)
Nelson, E. C., 711 (Dys.)
Neogi, S. K., 851 (Mal.)
Nesserlin, H., with Sicault, (777) (Mal.)
Neumann, H., with Schilling, Schreck & Kunert, 195, 664 (S.S.)
Neveu-Lemaire, M., 643 (B.R.)
Newman, M. S., with Anderson, Crowder & Stodola, 940 (Lep.)
Ngo-quang-Ly, with Dorolle, 309 (Lep.)
——, with ——, Huynh-van-Huy & Tran-van-Tam, 612 (Lep.)
Nguyen-Dinh-Hao, with Morin & Gaschen, 836 (Mal.)
Nguyen Ngoc Nhuan, with Montél, Bablet & Do van Hoanh, 944 (Lep.)
——, with ——, Le-van-Phung, Nguyen-van-Khai, Tran-van-Hanh, Do-van-Hoanh & Truong-van-Que, 309 (Lep.)
Nguyen-van-Huong, with Ragiot, Delbove & Ho-thieu-Hgan, 354 (Lept.), 433 (Fev.)
Nguyen-van-Khai, with Montel, Le-van-Phung, Tran-van-Hanh, Do-van-Hoanh, Fruong-van-Que & Nguyen-ngoc-Nhuan, 309 (Lep.)
Nguyen-van-Luong, with Fréville, (769) (Mal.)
Nguyen-van-Tan, with Bourgin (228) (Mal.)
——, with Massias & Bourgin, 271 (Mal.)
Nguyen-van-Tung, (312) (Lep.)
Nicholls, L., 65 (Sp.)
Nicol, W. D., 279 (Mal.)
——, with James & Shute, P. G., 684 (Mal.)
Nicolau, S. & Baffet, O., 322 (Rab.)
——, Mathis, M. & Baffet, O., 622 (Y.F.)
Nicoletti, F., 965 (Y. & S.)
Nicolle, C., 438 (Fev.)
—— & Laigret, J., 34, 437 (Fev.), 619 (Y.F.)
—— & Sparrow, H., 44 *bis*, 48 (Fev.)
Nieuwenhuis, A. W., 754 (Mal.)
Nieva, D. E., 95 (Hel.)
Nigeria, 172 (S.S.), 337 (Y.F.)
Nigg, C., 424, 916 (Fev.)
Niimi, S., 210 (S.S.)
Nikolić, M., 325, 326 (Rab.)
Niño, F. L., 471 (Misc.), 591 (Hel.)
Nishiyama, K. & Nagahama, M., 965 (Y. & S.)

van Nitsen, R., (229) (Mal.)
Niven, J. C., with Field, 798 (Mal.)
Nobrega, P., with Reis, J. & Reis, A. S., 643 (B.R.)
Nocht, B., (613) (Lep.)
—— & Mayer, M., 883 (B.R.)
—— & Velasco, F., 315 (Lep.)
Noël, G., with Dubois, 439 (Fev.)
Nolasco, J. O., 296, (609) (Lep.)
Nolf, L. O., & Edney, J. M., 122 (Hel.)
Noosten, H. H. & Visser, J., 805 (Misc.)
Nouguès, S., with Nattan-Larrier, 28 (K.A.)
Noury, M., 430 (Fev.)
——, with Blanc, 699 (R.B.F.), 921, 924 (Fev.)
——, with —— & Baltazard, 437, 924 (Fev.)
Novák, E., with von Jancsó, 56 (R.F.)
Nukada, M., with Schöbl, 372 (Chl.)
de Nunno, R., 236 (Mal.)

O

Ochoterena, I., 103 (Hel.)
O'Connor, F. W., 526 (Am.)
—— & Hulse, C. R., 592 (Hel.)
O'Connor, M. P., 420 (Fev.)
d'Oelsnitz, 25 (K.A.)
Oesterlin, M., 663 (S.S.)
O'Flynn, E. G., with Pasricha & de Monte, 866 (Chl.)
Ogata, N., 441 (Fev.)
Ogilvie, W. H., 572 (B.R.)
Oguro, T., 537 (Am.)
Ohashi, K., 87 (Hel.)
Olavarria, J., with Hill, 270 (Mal.)
Olesen, R., 324 (Rab.)
Olmer, D., Olmer, J. & Audier, M., 910 (Fev.)
Olmer, J., with Olmer, D. & Audier, 910 (Fev.)
O'Meara, F. J., 447 (Fev.)
Orenstein, A. J. & Gordon, A., 567 (B.R.)
Orr, H. C., 905 (Oph.)
Osborn, H. A., 136 (Hel.)
Osburn, H. S., with de Meillon, 487 (Misc.)
Osgood, E. E., 483 (Misc.), 856 (Mal.)
Otten, L., 365 (Pl.)
Otto, G. F., 562, 563, 581, 588 (Hel.)
Otto, I. H., 523 (Bl.)
Ovchinnikov, M. M., with Rybinsky, 838 (Mal.)
Ozeki, Y., 59 (R.B.F.)

P

Pai, H. C., 803 (Misc.)
Pai, N. G., with Krishnan, 517 (Bl.)
Palacios, L. D., with Earle & Arbona, 837 (Mal.)
Palamedi, B., with Mazza, (678) (S.S.)
Palestine, 466 (Misc.), 749 (Rab.)
Palthe, P. M. van W., 74 *bis* (Misc.)
Panayotatou, A., 500 (K.A.), 537 (Am.)
Pandit, C. G., with Wright & Seetharama Iyer, 127 (Hel.)
Pandit, S. R., 777 (Mal.)
Panja, D., 407 (Diet.)
——, with Chopra & Chaudhuri, 145 (Der.)
Panja, G., (938) (Lep.)
Pannier, R., 672 (S.S.)
Papantonakis, E., 17, 500 (K.A.)
Paparcone, 449 (Oph.)
Pardal, E., 361 (Pl.)
Pardo-Castello, V., (605) (Lep.)
Pareli, A., (799) (Mal.)
Paris: Office International d'Hygiène Publique, (616) (Y.F.)
Parise, N., (721) (Misc.)
Parish, H. E. & Laake, E. W., 488 (Misc.)
Parker, R. R., 47, 440 (Fev.)
Parreiras, D., (870) (Pl.)
Parrot, L., with Sergent Edm., Sergent, Et. & Catanei, 757 (Mal.)
Parvis, F., with Scartozzi, 93 (Hel.)
Pasco, A. M., with Tubangui, Basaca & Del Rosario, 582 (Hel.)
Pascual, A., with Cano, (348) (R.F.)
Pasricha, C. L., Bannerjee, K. & Lal, S., 924 (Fev.)
——, de Monte, A. J. H. & O'Flynn, E. G., 866 (Chl.)
——, —— & Gupta, S. K., 865 (Chl.)
Passalacqua, R., with Uriarte & Argerich, 361 (Pl.)
Paterson, A. R., 251 (B.R.)
Paty, R. M., Jr., with Yui, (777) (Mal.)
Paul, M., 613 (Lep.)
Pavlinova, Y., with Taliaferro, 671 (S.S.)
Pavlovsky, E. N., 330 *bis*, 331 (B.R.)
Pawan, J. L., 227 (Mal.) 742 (Rab.)
Payne, G. C., with Castle, Rhoads & Lawson, 66 (Sp.)
——, with Gotay & Costa Mandry, 969 (Y. & S.)
Peck, S. M., Crimmins, M. L. & Erf, L. A., 395 (Sn.)
—— & Rosenthal, N., 394 (Sn.)
Pecori, G. & Escalar, G., 757 (Mal.)
Pedlow, J. T., with Reiner & Smythe, 670 (S.S.)
Peirce, E. R., 879 (Pl.)
Peirier & Raymond, 142 (Der.)
Peña Chavarría, A. & Rotter, W., 584 (Hel.)
Penfold, H. B., with Penfold, W. J. & Phillips, 564 (Hel.)
Penfold, W. J., Penfold, H. B. & Phillips, M., 564 (Hel.)
Pennanéac'h, J., with Le Chuiton & Berge, 439, 927 (Fev.)
——, with ——, Mondon, Berge & Dubreuil, 438 (Fev.)
Penso, G., 122 (Hel.), 533 (Am.)
Pepeu, F., 379, 397, 398 (Sn.)
Pereira, L., 934 (Lep.)
Pereira da Silva, E., 748 (Rab.)
Pereira, P. C. R., 942 (Lep.)
Perez, A., with Marañon & Russell, 232 (Mal.)
Pergher, G., 923 (Fev.)
Pergher, J. & Casier, J., 423 (Fev.)
Peristeris, M., with Foy & Kondi, 793 (Mal.)
Perla, D. & Marmorston, J., 163 (B.R.)
Perry, H. M. & Poole, L. T., 489 (Misc.)
Peruzzi, M., 183 (S.S.)
Peschle, B., 444 (Fev.)
Peter, F. M., 233 (Mal.)

Peters, B. G., 947 (Hel.)
Petersen, M. C., 856 (Mal.)
Petrov, V. B., 25 (K.A.)
——, with Chodukin & Kevorkov, 500 (K.A.)
Petruschevsky, G. K. & Boldyr, E. D., 98 (Hel.)
Pflomm, E., 159 (Misc.)
Phillips, M., with Penfold, W. J. & Penfold, H. B., 564 (Hel.)
Piéri, J., 26, 502 (K.A.)
—— & Sardon, 952 (Hel.)
Pieter, 606 (Lep.)
Pietermaritzburg, 422 (Fev.)
Pile, G. R., with van Rooyen, 279 (Mal.)
Pillai, M. J. S. & Murthi, K. N., 65 (Sp.)
Pinkerton, H., 907 (Fev.)
Pinto, G. de S., (269) (Mal.)
Piot, M., 633 (Misc.)
Pirumow, 854 (Mal.)
Pitruzzella, R., 504 (K.A.)
Pittaluga, 499 (K.A.)
Piza, J. de T., 40 (Fev.)
Plantilla, F. C., 604 (Lep.)
——, with Doull, Rodriguez & Guinto, 931 (Lep.)
—— & Mabalay, E., 402 (Sn.), 966 (Y. & S.)
——, with Rodriguez, 604 (Lep.)
Plazy, L. & Germain, A., 41 (Fev.)
Plotnikov, N. & Zertchaninov, L., 559 (Hel.)
Plotz, H. & Giroud, P., 916 *bis* (Fev.)
——, with ——, 42, 43, 438 (Fev.)
Plum, D., 115 (Hel.)
Poggi, I., (113) (Hel.), 257, (290) (Mal.), (636) (Misc.)
Poindexter, H. A., 77 (Misc.), 184 (S.S.)
Pokrowskaja, M., 876 (Pl.)
Pokrowski, S. W., 283 (Mal.)
Polidori, T., 721 (Misc.)
Pollitzer, R., with Wu Lien-Teh, Chun & Wu, 726 (B.R.).
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Printed under the authority of His Majesty's Stationery Office
by the South Essex Recorders, Ltd., High Road, Ilford.

(2267) Wt. P3312A/183 1875 6/37 S.E.R. Ltd Gp. 353.